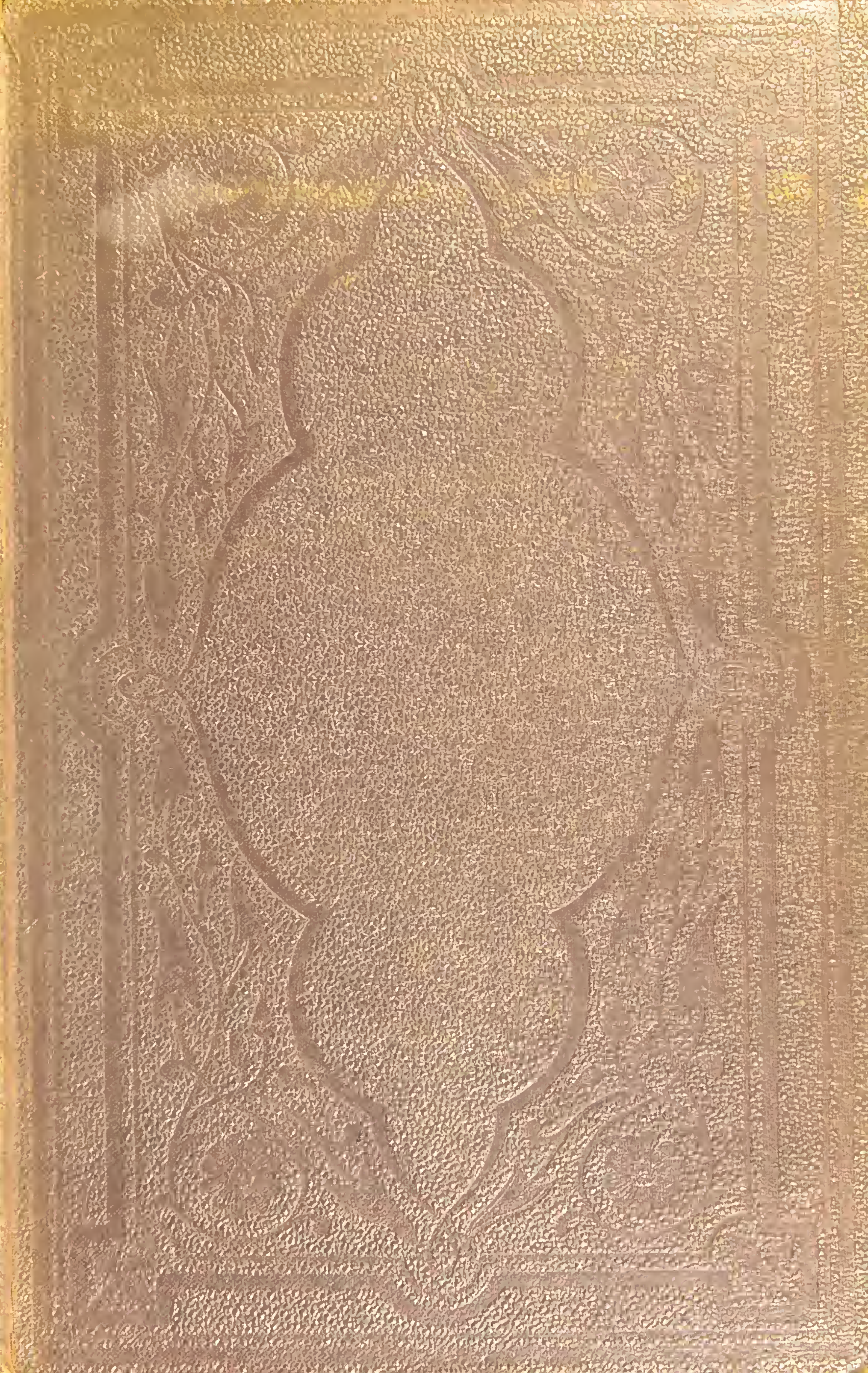

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THE

DISEASES OF THE STOMACH,

WITH AN

INTRODUCTION ON ITS ANATOMY AND PHYSIOLOGY.

24. i. 25,

LECTURES

ON THE

DISEASES OF THE STOMACH,

WITH AN

INTRODUCTION ON ITS ANATOMY AND PHYSIOLOGY.

BY

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SECOND EDITION.

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PREFACE.

The following Lectures are published, in the hope that they will be found a brief but complete account of what is at present known concerning the Diseases of the Stomach.

Fifteen years ago, the death of a near relative painfully impressed me with the obscurity and uncertainty which peculiarly attach to the maladies of the Abdominal Viscera. From that time I have given to these maladies whatever attention could be justifiably devoted to a special group of diseases by a student and practitioner of Physic in general.

Of the materials I have thus collected, those which form the basis of the following Lectures are derived from sources so various, as to render the concord equally important with the number, of the facts they represent. To the large opportunities afforded me, during twelve years, as a Dispensary and Hospital Physician, I have added whatever could be gleaned by me from a field of observation, the magnitude of which it is difficult to estimate. The Hospitals and Museums of various cities; the cases recorded by authors of various epochs and countries; and especially, the stores of information which are gradually accumulating in the Journals, Reviews, and Transactions of the Medical Profession in the more civilized parts of the world—have all been laid under contribution; and have yielded me facts which (considering the price of human suffering paid for Pathological knowledge) deserve a respect and attention I sincerely disclaim for the manner in which I have arranged and used them. To this general acknowledgment I must add my special thanks to numerous professional friends, and to hundreds of observers whose smaller contributions could not be separately quoted in the following Lectures.

Of the mode in which the subject is treated, it need only be said, that I have endeavoured to incorporate, with a clinical and pathological inquiry into gastric disease, such physiological principles as may afford a basis for its rational treatment.

In making this attempt with the brevity necessary to bring it within the limits of a short course of Lectures, I am sensible that retrenchment may have been misapplied, or carried too far. But for the latter fault, the reader will, perhaps, find excuses more valid than I can offer. It may, however, be mentioned, that my audience was composed chiefly of the more advanced students of St. Thomas's Hospital; who, having attended the excellent Clinical and Systematic Lectures on Medicine of my colleagues (Dr. Barker and Dr. J. R. Bennett respectively), had already an elementary acquaintance with my subject.

Perhaps, besides the defects thus hinted at, I ought also to apologise for redundancies (as in Lectures III. and VI.) But, apart from the special connexion of Therapeutics with gastric disease, I have a conviction that, in the present era of the Art of Medicine, the language of those who teach it should at least be outspoken, if it may not be eloquent in the more conventional sense of this word.

The Lectures thus delivered have received some additions and corrections in passing through the press; and have been increased in number by one (Lecture V.) which was read as a paper before the Physical Society of St. Thomas's Hospital during the present Session. For the convenience of the reader, I have prefixed a summary of the Anatomy and Physiology of the Stomach. This chapter, derived from my Physiological Lectures, is illustrated by some engravings, drawn on the wood by myself.

WILLIAM BRINTON.

BROOK STREET, GROSVENOR SQUARE,
December, 1858.

PREFACE TO THE SECOND EDITION.

In preparing this Edition for the press, I have taken the opportunity of revising and enlarging it. Of the additions thus made, many are dispersed throughout the body of the work. I have, however, added two chapters; the clinical interest of which will, I hope, justify their admission to what still only claims to be a brief account of the Diseases of the Stomach.

WILLIAM BRINTON.

24, Brook Street, Grosvenor Square,
October, 1864.

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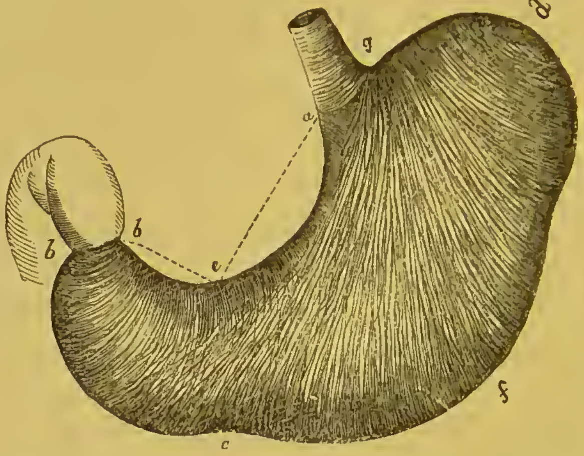
ANATOMY AND PHYSIOLOGY.

The stomach, the widest and most dilatable part of the alimentary canal, has a form which varies somewhat in different individuals. Removed from the body, and moderately distended, it generally takes the shape here represented (Fig.1)—a shape best described as that of a bent cone, the concave aspect of which receives a tube at one fourth of the distance from its base. In it we distinguish an anterior and a posterior surface; a superior and an inferior border; a right and a left extremity; and lastly, the cardiac and pyloric apertures, by which it communicates with the œsophagus and duodenum respectively, and thus becomes continuous with the remainder of the digestive canal.

The description of these parts varies with the state of the organ. Thus, when empty and uncontracted, the stomach is flattened vertically; its anterior and posterior surfaces touching each other, while its upper and lower margins really deserve the title of "borders." But when the organ is distended, any transverse vertical section becomes almost a circle, its borders and surfaces merging into each other. Its uppermost part, however, is still distinguishable as the *lesser curvature* (*a*, *e*, *b*, Fig. 1), and its lower as the *greater curvature* (*g*, *d*, *f*, *c*, *b*). The general concavity of the former curve is especially marked in its first three-fifths, at the end of which part (*e*) it usually becomes slightly convex. A shallow notch (*c*) often divides the greater curvature into two portions opposite this point, and, with the latter, defines the commencement of the pyloric pouch (*c*, *b*, *b*, *e*). The *cardiac pouch, great or splenic extremity* (*d*) is the part to the left of the *cardia* or œsophageal opening (*a*), beyond which it projects for about three inches. At this aperture the œsophagus dilates gradually, so as to resemble an inverted funnel. To the right of the œsophagus, the stomach expands slightly, to reach its maximum diameter at about the

FIG. 1.



Stomach, as seen by inflating it, and dissecting off its peritoneum, its longitudinal, and part of its transverse, muscular coat.

a, g, Cardia; *b, b,* pylorus; *a, e, b,* lesser curvature; *g, d, f, c, b,* greater curvature; *g, d,* to near *f,* cardiac sac; *c, b, b, e,* pyloric sac. (Above *a, g* are seen the transverse fibres of the œsophagus; and below these, the uppermost of the oblique fibres of the stomach, passing towards *c*. Covering the pyloric sac are seen the transverse fibres. The dotted line, *a, e, b,* shows how extreme distension of the stomach tends to affect the lesser curvature.)

middle of the organ (*f*). Beyond this point it tapers away to the *pylorus* (*b, b*), where a sudden constriction marks the site of the valve.

The dimensions of the organ are even more variable than its form. In the healthy middle-aged male, the moderately distended stomach is about thirteen to fifteen inches long; and its diameter, at the widest part five, near the pylorus two, inches. Its total surface is about one and a quarter square feet; its capacity about 175 cubic inches, or five pints; its weight seven ounces.[1]

The attachment of the stomach is effected chiefly by the continuity of its extremities with the more fixed duodenum and œsophagus. The former tube is connected with the posterior wall of the belly; the latter perforates the diaphragm, so as to enter the abdomen about one inch in front of the left border of the aorta, by an aperture which is everywhere muscular, though close to the posterior border of the tendon. The fixation of the stomach is also aided by certain processes of peritoneum. To the left of the œsophagus, the short *phreno-gastric omentum* passes from the diaphragm to the cardiac pouch, which it reaches somewhat posteriorly. Still lower down, the stomach is united to the spleen by the *gastro-splenic omentum.* The lower border of the organ gives off the *great omentum ;* this descends for some distance towards the bottom of the belly, and is then reflected upwards to the anterior border of the transverse colon, which it splits to enclose. The upper border of the stomach is attached by means of the *gastro-hepatic* or *small omentum,* which descends from the transverse fissure of the liver. All of these folds are double; though the four layers of the reflected *omentum majus* are often inseparably united to each other.

---

[1] For women and children, these estimates require a proportionate reduction. They are increased by habitual distension, and by the relaxation of old age; diminished by habitual exercise, or by the practice of taking very small meals (as in dilative emphysema of the lungs).

Situation.—The stomach is placed almost transversely in the upper part of the abdominal cavity, in which it passes from the left to the right side, as well as downwards, and slightly forwards. This direction results from its situation relatively to the œsophagus and duodenum; since it is joined by the former at its highest part, and near its left extremity, while the latter is immediately prolonged from its right or pyloric end. In this course from left to right, the stomach successively occupies the left hypochondriac and the epigastric regions; and, just at its termination, it reaches the right hypochondrium. Its anterior surface is therefore in contact with the diaphragm, where this muscle lines the cartilages of the left false ribs, and with the wall of the abdomen. Its posterior surface lies upon the pancreas, the aorta, and the crura of the diaphragm, where these parts cover the spine. Its left extremity is in contact, above, with the diaphragm; below with the spleen; and, posteriorly, it reaches the left supra-renal capsule and kidney. Its upper border is in apposition to the liver; viz., to its left lobe, to the *lobulus Spigelii,* and to part of the *lobulus quadratus.* Its lower border is parallel, and close to, the transverse colon.

Unusual distension or size chiefly affects the situation of the organ, by causing it to extend downwards, so as to overlap or displace the transverse colon, and thus to reach the umbilical, the left lumbar, or even the left iliac region. Under similar circumstances, its left extremity also passes more deeply into the corresponding hypochondrium, so as to be more extensively covered by the ribs. Its extension upwards diminishes the size of the thorax, but is rarely sufficient to be felt as a serious hinderance to the descent of the diaphragm in ordinary tranquil inspiration. Its right extremity may touch the gall-bladder.

It may be useful to trace the effect of progressive distension of the stomach upon its form, site, and fixation. When void of food, and not distended (as it often is) by gases, the flattened stomach hangs almost vertically in the epigastrium. In this state of the organ, the pulpy food that enters it from the œsophagus drops at once into the cardiac pouch, which forms its most depending part. The reception of further quantities effaces its upper and lower borders; and gradually changes them, from almost straight lines, into the curves above mentioned, at the same time that it separates the previously apposed surfaces, and converts the whole organ into a cone, convex below and in front. The latter of these two convexities is most marked at the pyloric extremity, and is often very sudden. Both result from the increased length of the organ, and the proximity of its comparatively fixed orifices. But both are greatly assisted by the muscular coat; since the distension of the separated stomach tolerably imitates (though it scarcely equals) the curves taken by the organ when moderately expanded *in sitû*. The delicate and yielding *omenta* just enumerated allow the stomach to expand between their elastic and extensive laminæ, without undergoing any disturbance of its nervous and vascular connexions, or any loss of its serous covering. Finally, although the stomach itself enlarges pretty equally in all directions, still, after filling the left hypochondrium, the mobility of its bent middle directs it towards that part of the enclosing cavity where it meets with the least resistance—namely, towards the yielding anterior wall of the belly. Hence, should the distended intestines not allow it any great descent downwards, it comes forwards; so that what was its vertical surface now looks obliquely upwards, while its inferior border touches the lower part of the wall of the epigastrium, where its artery has even been felt pulsating in very emaciated subjects.

In the physical examination of the stomach during life, the accuracy with which the outline of the organ can be mapped out by percussion is, of course, subject to great variations. If tolerably distended, the mere tone afforded by the cardiac end will generally distinguish it from the colon (and *a fortiori* from the small intestine), no matter how great or irregular the flatulent distension of its adjoining segments. The gradual merging of this deep tympanitic sound into that of intestine, near the pylorus, is a source of less ambiguity than might be expected; both from the proximity of this part to the surface, as well as from its marked contrast with the liver and colon above and below respectively. Often, however, the latter aid to distinction is absent, from the small or large intestine overlying the stomach for a longer or shorter period; a state which is, I think, more frequently produced by the segment thus concealing this stomach being pushed over by the distension of adjacent parts, than by its own flatulent dilatation. Lastly, a distended cardiac sac can scarcely be outlined with exactness; its tympanitic resonance being so gradually obscured by the increasingly thick wedge of lung which intervenes between the hypochondriac integuments and the highest (or diaphragmatic) part of this sac, that no definite line of demarcation is generally traceable.

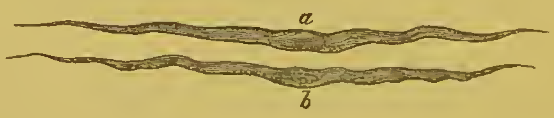
In common with all the sub-diaphragmatic segments of the alimentary canal, the stomach is composed of three coats or tunics:—an external and serous, a middle and muscular, and an internal and mucous coat. The first of these attaches the organ to the cavity in which it is enclosed; and limits, permits, and facilitates those movements which it is the chief office of the second to execute. The third is the most important, forming the secreting and absorbing surface on which the functions of the organ chiefly depend.

The *serous* coat of the stomach is continuous with the double laminæ of peritoneum before mentioned, which split to enclose it where they reach its various borders. Here they are very loosely connected to each other, and to the subjacent coat, by an abundance of highly elastic areolar tissue. But towards the middle of the gastric surface, the peritoneum, though still elastic, is closely united to the subjacent muscular tunic. The advantage of this mode of attachment has already been referred to.

The structure of the serous coat is precisely that of the visceral peritoneum elsewhere. A single layer of flattened epithelia, of hexagonal shape, rests upon a stratum of areola tissue, containing the scanty vessels by which this cell-growth is nourished. Immediately beneath the epitheliar layer, the areolar tissue is condensed and firm; and its aspect in contact with the cells shows the smooth continuous outline of what is, developmentally, a basement membrane, but is actually inseparable from the fine yellow fibres beneath, through which it gradually merges into loose, elastic, sub-serous tissue. The large meshes of the latter often enclose variable quantity of adipose tissue.

The *muscular* coat of the stomach consists of the unstriped or organic muscular fibre, which Koelliker has shown to be constructed of fibre-cells (Fig. 2, *a*, *b*). The length of these cells is from $\frac{1}{200}$th to $\frac{1}{100}$th of an inch; their breadth from $\frac{1}{6000}$th to $\frac{1}{4000}$th at the middle, where they are flattened, and from whence they taper off to conical and pointed extremities.

FIG. 2.



Fibre-cells of the human stomach, magnified 450 *diameters.*

a, Fibre-cell, as usually seen; *b*, fibre-cell, with wavy, irregular edge.

They contain a nucleus, which is from $\frac{1}{2000}$th to $\frac{1}{1000}$th of an inch in length, and about a sixth of this in breadth. Their texture is a pale substance, apparently homogeneous, but consisting, in reality, of a membrane enclosing granulated or faintly striated contents. In other instances they are marked by swellings (as in *b*), which, as they are rarely seen in the associated fibres, are probably due to casual local contractions of the sarcous substance itself. The arrangement of these fibre-cells is very simple; they are packed together in parallel rows, their flattened surfaces adhering strongly to each other. They thus form small bundles, between which are interposed the vessels for their supply, enclosed in a sparing quantity of areolar tissue. The union and interlacement of these fascicles build up the strata of the muscular coat. The development of the fibre-cell takes place by the elongation of an oval cell, in which, at the same time, is deposited a sarcous content, that soon obscures the original cell-membrane.

In the intestine these fibres are arranged in two layers—an external, in which the bundles take a longitudinal course; and an internal, in which they are circular or transverse[1] to the axis to the tube. But, in the stomach, the peculiar shape of the organ is associated with a modification of this arrangement.

The *longitudinal* layer of the stomach is derived from the similar tunic of the œsophagus. This tunic, on reaching the cardia, radiates on all sides; its bundles becoming thinner as they diverge, to be gradually lost among the various fibres with which they decussate and interlace. But, on the lesser curvature of the organ, they continue much more distinctly; and are often traceable, as two or three broadish bundles, to within a short distance of the pylorus. The longitudinal layer which covers the pyloric extremity appears not to have any very direct continuity with the preceding. Its constituent fibres arise by scattered bundles at about the middle of the organ, and—often first uniting into two broad bands which occupy the middle of its anterior and posterior surfaces—they soon form a tubular layer, which proceeds over the pylorus, to join the commencement of the duodenum.

The *transverse* or *circular* fibres lie immediately beneath the preceding, and form a much thicker layer. To the left of the cardia, its rings are very few and indistinct; their places being taken by those of the third or oblique layer. But from the right of this orifice, it continues towards the pylorus (*p*, Fig. 3), with a constantly increasing thickness; until finally, reaching the margin of this valve, it is inflected towards the axis of the stomach by a rather steep or sudden curve, which presents an almost vertical surface towards the duodenum. Those of its fibres which lie nearest to the left extremity are somewhat less regularly transverse. Hence some of them decussate slightly with each other; while others, which pass downwards from the right margin of the cardia, are directed somewhat obliquely towards the left extremity of the organ.

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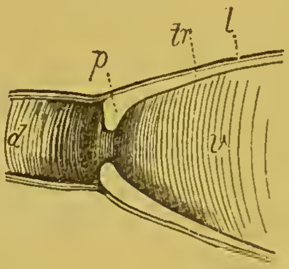
[1] Probably in reality spiral. For the grounds of this conclusion, see the author's Essay, "Stomach," *Cyclopædia of Anatomy*, Suppl., p. 311.

FIG. 3.



Longitudinal section of the stomach and duodenum, to show the pyloric valve.

v, Pyloric sac of the stomach; *l*, its longitudinal muscular coat; *tr*, its transverse one, gradually thickening into *p*, the pylorus; *d*, commencement of the duodenum.

The third or *oblique* layer lies more deeply than the two preceding; and is therefore best seen by everting and inflating the stomach, and carefully removing its mucous membrane. Where the œsophagus enters the stomach, the transverse fibres of its left margin are so close to a flattened bundle of fibres, which occupies the notch (*g*, Fig. 1) limiting the cardiac pouch, that the two are visibly continuous. The right or thickest part of this flattened band passes obliquely downwards towards the right side, soon breaking off from the termination of the œsophagus; and from hence it continues across the transverse layer just described, to reach the greater curvature, where the similar layers from both surfaces of the organ are reflected into each other. Its usually well-defined margin occupies—and indeed forms—the notch (*c*, Fig. 1). The posterior or thinner part proceeds, not only from the depression (*g*, Fig. 1) on the left of the cardia, but also from the neighbouring upper borders of the great extremity; and its more vertical fibres are also continued downwards to the lower border of the stomach, where they meet so as to complete the circuit of the cardiac pouch.

Movements of the Stomach.—That there is an intimate connexion between the œsophageal and gastric movements, is only what might be expected from that visible continuity of their muscular coats which has just been alluded to.

At the close of each act of deglutition, the lower fibres of the œsophagus contract with such force, as not only to obliterate the cardiac aperture, but even to cause the mucous membrane of this part to project into the cavity of the stomach. This condition remains during some moments. And when the alimentary bolus has thus been impelled into the organ, it excites muscular movements. As regards the state of the cardia during stomach-digestion, the force with which it is shut is effectively superior to the pressure exerted on the contents of the organ by the gastric contractions. Still, we are ignorant how much of this force is due to the contraction of the lower œsophageal fibres, and how much to the shape, position, or structure of the stomach itself. But it is probable that the decussation of the transverse and oblique fibres of the organ around the insertion of the œsophagus, renders their contractions a material assistance to the obliteration of the lower part of this tube. In any case, there can be no doubt that the cardiac orifice is chiefly closed by an active muscular contraction of its own muscular walls :—a contraction which is excited by the stimulus of food in the stomach, and is independent (so far as can be judged) of all assistance from the diaphragm.

The movements executed by the stomach, and impressed upon the food during its sojourn in this cavity, vary according to the stage of digestion, and the nature of the aliment.

The empty fasting stomach offers no movement whatever. And an unnaturally solid or massive food is not only incapable of movement within the organ, but embarrasses or checks the action of the muscular coat. A small quantity of liquid food excites a vermicular action, a gentle contraction or grasping motion of the stomach, so that the wrinkles of the mucous membrane gently close upon it, and gradually diffuse it over the whole surface.[1]

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[1] Beaumont. "Experiments and Observations on the Gastric Juice," Combe's Edition, 1838.

But the ordinary state of the human stomach during its digestive act lies between these extremes; and may be defined as one of moderate distension, with food which has been subdivided by mastication, and diluted with saliva and gastric juice, so as to possess a pulpy or semi-fluid consistence.

Shortly after the ingestion of such food, the stomach becomes the seat of a peristalsis or transverse constriction, which starts from the cardia, and travels thence slowly towards the pylorus. Comparatively feeble until it reaches the commencement (*c*, *e*, Fig. 1) of the pyloric sac, it here becomes much more distinct, and continues rapidly forwards as a well-marked circular depression, until it reaches the firmly shut pylorus. Its arrival here is followed by a relaxation; interrupted, after lasting about a minute, by a repetition of this peristalsis—which, by the way, is accompanied by some longitudinal shortening of the stomach.

As respects the movements which such contractions impress upon the food, there is the closest correspondence between the mechanical consequences of the process above described, and the results actually observed. The effect of peristalsis in a closed and distended tube may be represented by an inflexible hollow cylinder (Fig. 4), filled with liquid, and accurately fitted with a perforated septum, which is capable of free movement along its interior. Let such a septum be moved in either direction, and it at once exerts a pressure on the body of liquid contained in that end towards which its motion sets. The pressure being equal in all directions, a portion of the fluid escapes backwards through its aperture. This retrograde current will be continually lengthened by the advance of the septum along the

FIG. 4.

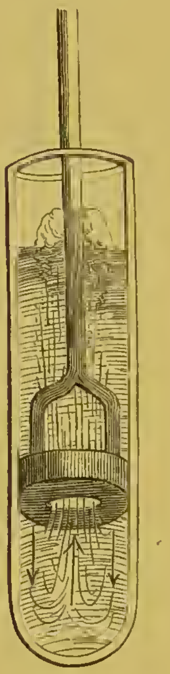

Diagram to illustrate the effect of peristalsis in a closed tube containing liquid.

The perforated septum is supposed to be moving downwards, causing the two currents (the peripheral downwards, the axial upwards) indicated by the corresponding arrows.

remainder of the tube. And the slow successive movement of a series of such septa would thus establish two continuous currents in the liquid—a peripheral of advance, and a central of return.

The existence of two such currents is little affected by the membranous nature and peculiar shape of the human stomach (Fig. 5). For, just as the comparative inactivity of the cardiac pouch would not prevent their occurrence as a consequence of pyloric peristalsis, so the very moderate contractions of this sluggish part suffice to define the axis and its current, as that curved line which unites the cardiac and pyloric apertures. "The bolus of food, as it enters the cardia, turns to the left, passes the aperture, descends into the splenic extremity, and follows the great curvature towards the pyloric end. It then returns in the course of the smaller curvature, makes its appearance again at the aperture in its descent into the great curvature, to perform similar revolutions. These revolutions are completed in from one to three minutes."[1] In other words, there is a backward movement of the gastric contents, from the pylorus, along that real axis of the stomach which unites the two orifices of the stomach near its lesser

FIG. 5.

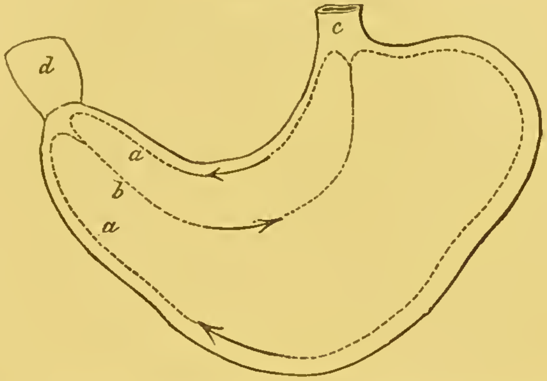

Diagram to show the movement impressed on the food in the stomach.

a, a, The peripheral or surface current effected by peristalsis, carrying the semifluid food towards the closed pylorus; where it is reflected into *b*, the central or axial current, occupying that real axis of the stomach, which unites its cardiac (*c*) and pyloric (between *b* and *d*) apertures.

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[1] Beaumont, *op. cit.*, p. 101.

curvature. Thus every part of the stomach is occupied by one or other of the two currents; and the mutual interference of these at their borders, gradually causes a uniform diffusion of the various alimentary matters moving with them. Finally, the reflexion of one current into the other at the pylorus, ensures an equal contact of all the semi-fluid food with the surface of the mucous membrane; since those portions of the food which occupy the axis of the stomach during one moment, are destined to move along its periphery during the next.

At a later period of gastric digestion, the contractions of the cardiac extremity appear even less active than before; and the longitudinal shortening of the organ is also less marked. The chief visible commencement of contraction is at the same place (*c, e*, Fig. 1), where it was formerly increased, and where it now forms a deep constriction or hour-glass contraction. After this constriction has continued a short time, it sends onwards towards the pylorus a rapid peristalsis, which appears nearly to obliterate the tube in its course, and ends by engaging the muscular ring of this valve. A slight relaxation closely follows this peristalsis, and is succeeded by a complete dilatation of the pyloric sac. Lastly, the hour-glass contraction itself sometimes disappears; and an interval of about two to three minutes precedes the repetition of the whole process. Often, however, the constriction remains until the peristalsis recommences.

The effect of these contractions on the contents of the stomach is twofold. The obliterative peristalsis of the pyloric sac arrests all axial current here, and extrudes a small portion of its contents into the duodenum. But in the intervals of these violent contractions, the ordinary peristalsis appears to be attended by the double current above alluded to.

The structure of the pylorus already described, and the movements of the stomach just specified, demand a very different view of its action than that selective power usually attributed to it, and implied by its very name (πυλωρός, *portæ custos*). Far from being a specific and independent structure, which contracts against the food in the earlier stage of digestion, but subsequently relaxes to permit the passage of the chyme, it must be regarded as a mere terminal thickening

(Fig. 3, *p*) of the transverse coat; with a strength proportional to its bulk, and an office, not only closely analogous[1] to that of the transverse fibres, but almost identical in both stages of gastric digestion. Instead of relaxing only at the end of this act, to allow a moderate peristalsis to urge through its aperture a selected portion of food, the pylorus is, at all periods of stomach-digestion, a contracted inflection of the transverse coat; through which the more fluid and homogeneous parts of the gastric contents are continually being strained, in small quantities, and at frequent intervals, by a more or less violent muscular effort—by a process, in short, of coarse filtration, aided by mechanical pressure.

Mucous Membrane.—The mucous membrane, on which the functions of the various parts of the intestinal canal essentially depend, is so modified in the stomach, as to offer a complex arrangement, such as remarkably contrasts it with the simpler layer that lines the pharynx and œsophagus. And it is distinguished from the compound membrane of the intestine by the possession of a special structure:—namely, the proper gastric cell, or glandular epithelium, as it is sometimes called.

The remaining histological constituents of this mucous membrane are similar to those met with in the intestine. A delicate membrane is involuted or moulded upon a quantity of areolar tissue. The latter texture thus forms the matrix of the mucous coat; and as such, contains its vessels, nerves, and lymphatics, and connects it with the middle or muscular coat. While, on its opposite side, the limitary or basement membrane sustains a number of minute cells, which bound the cavity of the canal.

Examined by the naked eye *in situ*, the mucous membrane of the stomach is seen as a tolerably firm but soft layer, which has a pale pink colour, and everywhere loosely lines the interior of the muscular coat, projecting from its surface in numerous wrinkled folds. These *rugæ* chiefly occupy the cardiac half of the organ, forming convolutions which, though somewhat irregular, are mainly longitudinal. They are effaced by distension of the stomach. On putting the mucous membrane on the stretch, we may often discern that its whole internal surface is occupied by extremely minute pits or depressions; the confluent and projecting intervals of which become so much longer as they near the pylorus, that they may be compared to short villi. These depressions are the openings of the stomach-tubes or proper gastric glands.

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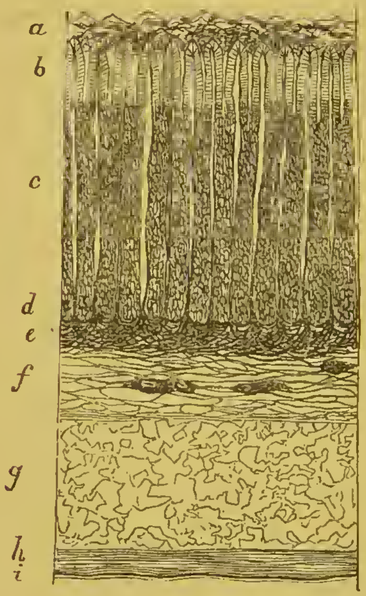
[1] It is interesting to observe how little the action of the pylorus is connected with any stimulus other than a gastric one. The flow of bile into the fasting stomach may perhaps be regarded as a transit, such as this janitor might well concede to a fluid which is not only harmless, but recrementitious. But in the obstructed canal, fæces pass through the valve from the duodenum with equal facility, although the stomach soon resents their presence by vomiting—an act which seems generally to imply a shut pylorus. And Magendie has observed, that the gases of this portion of the intestine can be made to pass the valve with equal facility; while those distending the stomach excite its contraction.

FIG. 6.



Vertical section of the stomach, near its middle, and parallel to its long axis. Magnified 30 *diameters.*

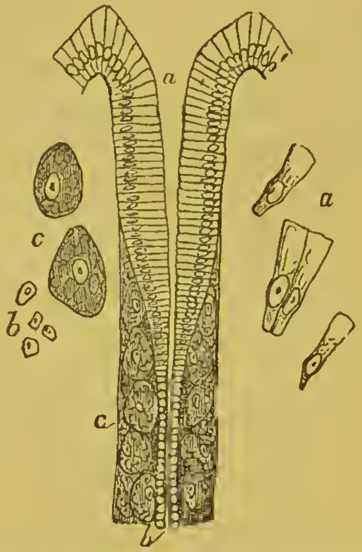
a, Openings of stomach-tubes, and their intervening ridges or projections; *b*, upper parts of the tubes, lined by columnar epithelium; *c*, lower parts, occupied by proper gastric cells; *d*, rounded ends of the tubes; *e*, dense areolar tissue, containing fibre-cells, and continuous with the intertubular matrix; *f*, submucous areolar or cellular coat, of a looser texture, and containing vessels (some of which are seen cut across); *g*, transverse layer of the muscular coat; *h*, longitudinal layer; *i*, peritoneal coat.

The *stomach-tubes* (*a, b, c, d*, Fig. 6) may be described as cylinders of basement membrane, which are packed vertically side by side in a sparing matrix of dense areolar tissue, and are filled by a peculiar cell-growth. Below, they terminate in closed and rounded extremities (*d*). Above, they expand slightly, before reaching the free surface of the membrane (at *a*); where their margins finally become continuous with each other, so as to form a series of low ridges, the height and width of which vary somewhat in different parts of the stomach. The length of these tubes is, on an average, about $\frac{1}{25}$th of an inch. Their diameter is about $\frac{1}{350}$th of an inch. Thus their length has to their breadth a proportion of ten or twelve to one. Their form frequently so far deviates from that of a simple cylinder, as to present slight constrictions or undulations. And occasionally they even exhibit a kind of cæcal pouch or blind offset of greater or less length. These pouches usually spring from the lower extremities of the tubes, which have generally a somewhat increased diameter in their neighbourhood. But with these exceptions (which are, I believe, the result of mechanical violence) the gastric tubes form simple, straight cylinders, and only widen where they open on the inner surface or cavity of the stomach.

The *limitary* or *basement membrane* of these tubes precisely resembles this delicate homogeneous layer in other mucous structures, possessing an equal (or even greater) tenuity. It is usually seen only as a dark outline, bounding some isolated part of a tube. Rarely, however, it may be identified as a delicate, floating, and collapsed fold, which, on the addition of a dilute alkali, first swells up, and then disappears. On the ridges which unite the tops of the tubes, it is quite impossible to separate it from the subjacent structures:—an intimate adhesion, in striking contrast to the ease with which we can often isolate it from the matrix around the tubes themselves.

As regards the *contents* of these tubes, the upper fourth or fifth of their length presents a single layer of columnar epithelium (*b*, Fig. 6; *a*, *a*, Fig. 7). Seen as isolated cells, the particles of this epithelium have a cylindrical shape, and enclose a very distinct nucleus near their attached extremity. But when seen in their natural situation, and from the free side of the mucous membrane, they appear as hexagonal prisms, containing nuclei, which are so near to their lower ends as to be separated from the basement membrane by little more than their cell-wall at this part. The remainder of the tube is under normal[1] circumstances always occupied by oval or somewhat angular cells (at *c*, Fig. 6; *c*, *c*, Fig. 7) of considerable size. The largest of these oval cells are about $\frac{1}{1200}$th of an inch in diameter. They have a more or less distinct membranous wall. The nucleus they contain is usually in contact with that side of their svarietes which is attached to the basement membrane of the tube; and it sometimes exhibits a nucleolus. Their contents are finely granular, with here and there refractile dots, which have a close resemblance to oil-globules. And, besides the above granular material, most of these cells appear to enclose numerous (5—15) pale, flat, and extremely delicate cytoblasts. The centre of the layer formed by these cells is apparently lined by a series of small angular cells (*b*, *b*, Fig. 7), which surround and enclose a narrow thread-like calibre or cavity; and above, merge into the columnar epithelium of the

FIG. 7.



Upper part of a tube from the middle of the human stomach, showing the arrangements of its columnar and oval cells. Magnified 200 *and* 500 *diameters.*

a, *a*, Columnar cells of the upper part, free and *in situ*; *b*, *b*, small angular cells, also free and *in situ*, into which these merge below, to form a central or axial layer within *c*, *c*, the proper gastric or glandular cells, also free and *in situ*.

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[1] In reference to the alleged expulsion of these during gastric digestion, compare the remarks on the gastric juice at the end of this chapter.

upper part of the stomach-tube. The interstices of the oval cells seem to be occupied by granules and minute cytoblasts.

Lenticular glands are also found in the stomach. As regards their shape, size, situation, and contents, they correspond with the follicles[1] or solitary glands of the intestine. Their number varies extremely. Sometimes it is impossible to find any. In other specimens, they are scattered more or less thickly throughout the whole organ. They are said chiefly to affect the lesser curvature; but I have seen them sown very plentifully over the pyloric region only. In children, they are rarely absent.

Matrix.—The cylindrical tubes of the stomach are united to each other, in their whole height, by a sparing quantity of a fibrous network or matrix; their blind ends also resting upon a layer (*e*, Fig. 6), continuous with that surrounding their sides. Near the free or cavitary surface of the stomach, this dark firm matrix is almost homogeneous. But in the deeper parts of the mucous membrane it is easy to distinguish, in addition to vessels, fibres which surround the tubes, and decussate with each other. This matrix, which also surrounds the intestinal tubes, and thus extends from the cardia to the anus, is composed of a variable admixture of areolar tissue with unstriped muscular fibres. The latter appear to pass, from the oblique and transverse layers of the muscular coat, through the areolar tunic, to reach the intervals of the stomach-tubes, in the form of bundles, which decussate at an acute angle. The action of these fibres is probably connected chiefly with the adjustment of the mucous membrane to the effects produced by the contractions of the proper muscular coat.

Areolar Tissue.—A layer of loose submucous areolar tissue (the *tunica nervea* of authors) connects the mucous and muscular coats. Seen in vertical sections (*f*, Fig. 6), its thickness is a little greater than that of the layer of matrix beneath the ends of the tubes. It is composed of the ordinary white and yellow fibrous elements; the filaments of the latter being chiefly of small size. Externally, it is firmly connected with the muscular coat, from which it receives fibres, and to which it sends off processes to form the septa of the muscular bundles. But more internally, where it approaches the fibrous matrix, its meshes are very large and loose; so as to allow the mucous membrane to be thrown into folds by the contraction of the muscular tunic. It contains the vessels, nerves, and lymphatics which supply the other coats.

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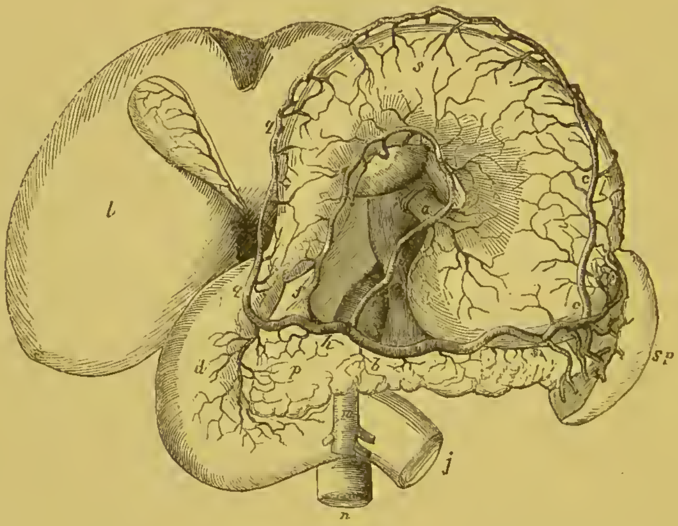
[1] See author's essay, "Intestine," *Cyclopædia of Anatomy*, Suppl., p. 357, *et seq.*

The *vessels* of the stomach are very large and numerous. The arteries are derived from the abdominal aorta. The veins empty themselves into the *vena portæ*, which ramifies in the liver.

The *arteries* come from the cœliac axis. This vessel, which leaves the aorta opposite the first lumbar vertebra, continues, obliquely forwards, as a short thick trunk, for about half an inch, when the "axis" ceases by giving off, at right angles to itself, three large branches—the gastric, hepatic, and splenic.

The *arteria coronaria ventriculi* (*a*, *a*, Fig. 8), or proper gastric artery, is the smallest of these three. It passes upwards and towards the left side, beneath the peritoneum which forms

Fig. 8.



Arteries of the stomach, as seen by raising this organ, to show their origin from the cœliac axis.

s, Stomach; *d*, duodenum; *l*, liver; *p*, pancreas; *sp*, spleen; *j*, jejunum; *a*, *a*, coronary artery; *b*, splenic artery; *c*, left gastro-epiploic artery; *e*, *vasa brevia*; *f*, superior pyloric artery; *h*, hepatic artery; *m*, superior mesenteric artery; *n*, aorta; *q*, *q*, right gastro-epiploic artery.

the dorsal and outward surface of the sac of the omentum; until, arriving nearly at the upper extremity of this cavity, it turns forwards in a slight projection or fold of the serous membrane. In this fold, it has a very brief and somewhat arched course, which brings it to the left end of the smaller curvature of the stomach. Here it passes between the two layers of the gastro-hepatic omentum. From hence it continues, in a very tortuous course, along this curvature: lying close to the stomach, and diminishing in size by giving off frequent branches; until, towards the right extremity of the organ, it becomes continuous with the pyloric branch (*f*) of the hepatic artery.

Its larger or named branches are the *œsophageal* and the *gastric.* The first are given off from the highest point of the vessel, or where it enters the gastro-splenic omentum. They run upwards to the œsophagus, taking a longitudinal course; so as to pass, with this tube, through the opening in the diaphragm, and anastomose with the thoracic vessels distributed to this tube from the aorta. The *gastric* ramifications run downwards from the artery on both surfaces of the stomach; inosculating, by their extremities, with branches from the splenic, gastro-epiploic, and superior pyloric arteries.

The *arteria hepatica* (*h*, Fig. 8), another branch of the cœliac axis, passes outwards and slightly forwards from the trunk to reach the commencement of the duodenum. It now runs almost vertically upwards, between the two layers of peritoneum that form the gastro-hepatic omentum, in front of the foramen of Winslow, to end by being distributed in the liver. In this course it gives off two branches to the stomach—the gastro-duodenal and the pyloric.

The *gastro-duodenalis* (beyond *h*, Fig. 8) is the first and largest of the two. It leaves the hepatic artery behind the duodenum, passing vertically downwards across the intestine to the lower border of its first portion. In this course it gives off a few small branches to the neighbouring parts of the stomach and intestine; some of which twigs have been distinguished as the *inferior pyloric* arteries. And at the inferior margin of the bowel, it bifurcates into two branches:—a large *gastro-epiploic* and a small *pancreatico-duodenal.*

The *gastro-epiploica dextra* (*g*, *g*, Fig. 8), the large vessel which continues the *gastro-duodenalis*, is so named from its course between those layers of the great omentum which descend from the stomach to form the "epiploon" or apron-like fold that covers the greater part of the intestinal canal. Beginning at the lower border of the duodenum, the artery runs from right to left, along the lower margin or great curvature of the stomach, and at a little distance from it, in wavy or tortuous curves. In this course, it gives off branches upwards to both surfaces of the organ; as well as others to the tissues of the omentum itself. And near the middle of the stomach, it ends by uniting with a corresponding branch, of smaller size, from the splenic artery.

The *arteria pylorica* (*f*, Fig. 8), which is sometimes distinguished by the title of the *pylorica superior* from the smaller branches of the gastro-duodenal above alluded to, is generally given off from the trunk of the hepatic artery opposite the upper border of the duodenum. More rarely it is derived from the commencement of its gastro-duodenal branch. In either case, it enters between the layers of the gastro-hepatic omentum, and runs in this fold, from right to left, along the upper margin or lesser curvature of the stomach, to join the coronary artery. It gives off numerous branches to both surfaces of the organ.

The *arteria splenica* (*b*, Fig. 8), the third branch of the cœliac axis, has no direct connexion with the digestive canal, until near its division into the terminal trunks by which it enters the spleen; where it gives off the left *gastro-epiploic* artery, and the *vasa brevia*.

The *gastro-epiploica sinistra* (*c*, Fig. 8) leaves the trunk of the splenic artery close to where this divides at the inner surface of the spleen. It passes downwards, forwards, and towards the right side; first lying for a short distance in the gastro-splenic omentum, and next entering between the layers of the great omentum continuous with this fold. It then runs along the lower border or great curvature of the stomach, to become continuous with the corresponding vessel on the right side, supplying branches to both surfaces of the stomach.

The *vasa brevia* (at *e*, Fig. 8) are small branches which come from the primary and secondary divisions of the splenic artery, and run in the gastro-splenic omentum to the cardiac pouch. Here they break up and anastomose with each other, as well as with the coronary and left gastro-epiploic arteries.

The *veins* of the stomach are the *superior pyloric,* and the *right* and *left gastro-epiploic.*

The *vena pylorica superior* receives and continues a large vein, which corresponds to the coronary artery, and takes a similar (but reversed) course along the lesser curvature of the stomach to the pylorus. It now passes upwards for a little distance, before opening into the *vena portæ* near its termination in the liver. In some instances, it bends down to join the splenic vein.

The *vena gastro-epiploica dextra* corresponds to the artery in the greater part of its distribution. It usually ends by emptying itself into the superior mesenteric vein, just before this forms the *vena portæ* by joining with the splenic vein.

The *vena gastro-epiploica sinistra* also runs with its artery, and joins either the splenic vein or one of its primary branches.

All of the foregoing vessels are characterized by the great freedom and frequency of their inosculations, in every stage of their course from the aortic to the portal trunks. This condition is especially well marked in the arteries, which in number and size far exceed those distributed to an equal bulk of most of the other structures of the body. This fact is doubtless connected, not merely with the large supply of blood they send to the stomach, but also with a smaller resistance, and greater velocity, in their channels; and especially with that efficient and sudden control of their calibre which the varying exigencies of the gastric circulation would seem to imply.

Their tortuous course, and their loose connexion with the stomach, chiefly refer to the distension of this organ. For as the stomach extends between the laminæ of peritoneum, it gradually straightens these vessels, and alters their position with respect to itself and each other.

The distal branches of both arteries and veins perforate the muscular coat at different intervals, by twigs which unite with each other in the loose submucous areolar tissue, so as to form two flattened networks:—one, which is composed of small arteries; and another, of veins. The vessels of the latter plexus are, as usual, both larger and more numerous than the corresponding arteries.

Capillaries.—The arterial branches which leave this submucous network to enter the dense muscular layer of the matrix of the stomach, divide here once or twice. And their ultimate ramifications, which have a diameter of about $\frac{1}{1500}$th to $\frac{1}{1800}$th of an inch, pass vertically upwards, along the sides of the tubes, to their upper apertures, where they form a superficial network of capillaries. In passing upwards, they also give off other capillaries; which surround the tubes at all parts of their height, so as to form a second and deeper network. The meshes of this latter plexus are somewhat oblong (*t*, Fig. 9), but less decidedly so than those of the capillary network of striped muscle; and are about $\frac{1}{100}$th to $\frac{1}{850}$th of an inch in size. The capillaries which compose them are, on a average, little more than $\frac{1}{5000}$th of an inch in diameter. The more superficial network (*r*, Fig. 9) is contrasted with this deeper one, not only in the fact that its capillaries are about double the above size (or $\frac{1}{2500}$th of an inch), but also in its meshes

FIG. 9.

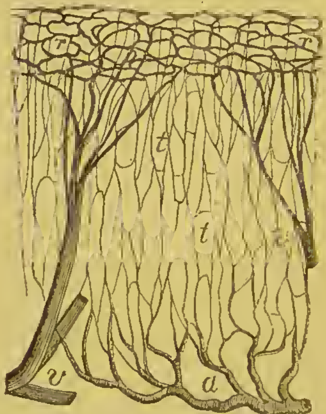
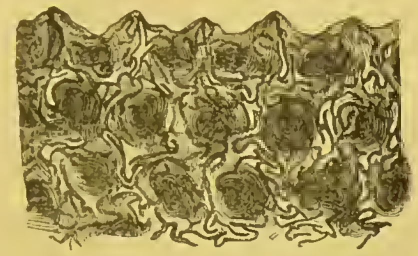


Diagram showing the vessels of the mucous membrane of the stomach, as they would be seen in a vertical section. Magnified 30 *diameters.*

a, Small artery of the plexus in the submucous areolar tissue; *t*, capillaries forming a network around the stomach-tubes; *r*, larger capillaries, forming a superficial network on the ridges which separate the open mouths of these tubes; *v*, veins, formed by branches from the latter network, and ending below in the submucous plexus.

(Fig. 10) being nearly twice as close (or about $\frac{1}{300}$th to $\frac{1}{800}$th of an inch). But the two plexuses inosculate so freely, as to be quite continuous with each other at the upper apertures of the tubes. As regards the form of the superficial network, it corresponds exactly with the intervals of the tubes. For the ridges which occupy the surface of the organ are all, as it were, moulded upon capillaries; the union of which forms a network that surrounds the aperture of each tube with a loop or ring (*r*, Fig. 9), complicated by the addition of other meshes (Fig. 10) on either side of it, just within the orifices of the tubes. In shape and size, these meshes closely resemble the loops beneath the ridges; and are, indeed, no way distinguishable from them except in their situation. Below, their diameter diminishes; their loops elongate; and they finally merge into the capillary network which surrounds the tube.

FIG. 10.



Superficial capillaries of the gastric mucous membrane, from an injected specimen. Magnified 60 *diameters.*

It is from the large capillaries which compose the superficial network that the radicles of the veins (*v*, Fig. 9) almost exclusively arise. They begin as small vessels of about $\frac{1}{1500}$th of an inch in diameter; and, by one or two successive unions of these and of their resulting larger branches, soon attain a width of about $\frac{1}{400}$th of an inch. They now pass vertically downwards between the tubes to open into the venous plexus of the submucous areolar tissue.

The general result of this arrangement on the circulation in the stomach is,—that the blood which has already traversed the capillaries of its tubes is further transmitted to its surface; and that, in respect to their size and situation, the superficial capillaries of the gastric mucous membrane offer some analogy to veins. Hence it is probable that the velocity of their contents far exceeds that of the blood circulating in the capillaries of many other tissues. Such a peculiarity would admirably adapt them to that absorptive office, which their situation on the cavitary surface of the stomach indicates to be one of their chief functions.

The *nerves* of the stomach are derived from two sources: from the pneumogastric, and the sympathetic, nerves.

The branches of the *pneumogastric* nerves leave these trunks at the lower end of the œsophagus; and, after a variable course in a nearly vertical direction—those of the left anteriorly, those of the right posteriorly, to the stomach—they perforate the muscular coat. Diminished in size, they then ramify in the submucous areolar tissue; in which they may be traced for a considerable distance, until lost from increasing minuteness on the under surface of the mucous membrane. In this course their complexity of arrangement almost baffles description. Each, for example, forms a network of its own branches near the cardia, as well as a larger plexus below this point. Each unites, not only with large and small branches of its fellow, but with the sympathetic nerves of the stomach, at all stages of their distribution visible to the naked eye—from the solar plexus and semilunar ganglia, to the secondary and tertiary offshoots of these around the vessels, and even to their branches in the areolar coat of the stomach.

The *sympathetic* nerves distributed to the stomach are branches from the great prevertebral centre of this nerve in the belly:—from the vast and complex ganglion formed in front of the aorta by the semilunar ganglia laterally, and the solar plexus which unites these in the middle line. The large plexiform ganglion thus constituted, gives off a process—the cœliac plexus—which envelopes the cœliac axis with a dense network of nerves; itself further breaking up into subordinate plexuses around the coronary, hepatic, and splenic branches of this arterial trunk (*a, b, h,* Fig. 8). Closely interlaced around these vessels, and doubtless from time to time distributing branches to their coats, the plexuses corresponding to the arteries just mentioned pass along them to the stomach; which they reach to penetrate the muscular coat, and to disappear in the sub-mucous tissue, like the gastric branches of the pneumogastric nerves. Of the three arteries, however, the coronary is that which appears to convey the greatest amount of nervous tissue to the stomach; and the pyloric branches of the hepatic plexus are next in number to these.

The further pursuit of all these nerves under the microscope adds little exact information to this description. Apparently, they interchange branches with each other at frequent intervals; though the arrangement is hardly of sufficient regularity and frequency to deserve the name of a plexus. Here and there indications of ganglion-corpuscles are sometimes discernible; as well as true bifurcations of the primitive nerve-fibres. And it is easy to verify a general diminution in the diameter of these fibres, in their course from the solar plexus to the under surface of the mucous membrane. But in this tissue itself, or between the secreting tubes of the organ, it has hitherto been impossible to detect nerves; and we can only conjecture their presence.

The functions of the nervous tissues derived from these two sources are also still uncertain. Section of both pneumogastric nerves remarkably interferes with gastric digestion: diminishing (if not almost suppressing) the secretion of gastric juice; provoking vomiting; and deranging (though not destroying) the due sensations of hunger and satiety. But where the animal survives, these effects seem to diminish, and even disappear. In like manner, violent irritation of the pneumogastric, or of the solar plexus, in a newly-killed animal, provokes movements in the muscular coat of the stomach. But such facts obviously prove little as to the exact offices of these nerves. And until we know far more of the real import of the numerous communications between the pneumogastric and sympathetic branches distributed to the stomach, a vague analogy to the corresponding arrangements of these nerves in the heart is almost all we can conjecture.

The *lymphatics* of the stomach consist of two sets:—one beneath the peritoneum, immediately outside the muscular coat; another (a plexus of larger vessels, with more frequent communications) in the submucous coat, between the mucous and muscular tunics. They anastomose freely at the ends of the stomach with the lymphatics of the liver, spleen, and pancreas; and are still more directly continuous with some glands, which are of small size in the healthy individual, and are placed chiefly near the lesser curvature of the organ. How far these vessels exercise the office of lacteals, in addition to that of absorbents—in other words, how far they are chyliferous as well as lymphatic—is a question which can hardly be answered in detail. But it is difficult to deny them some action of this kind, even while it is certain that they rarely, if ever, convey a visibly chylous liquid.

Changes during Digestion.—The inner surface of the healthy fasting stomach is of a pale pink colour. The mucous membrane itself, always exhibiting an acid reaction, is covered by a thin stratum of alkaline fluid, derived (as its chemical and microscopical examination abundantly proves) from the various secretions which enter the organ by the œsophagus and duodenum. The introduction of food gives rise to two chief alterations in the state of the stomach. Its muscular coat is excited to the movements already described. And at the same time, its mucous-membrane deepens to a bright pink colour, and begins to pour forth a liquid—the *gastric juice.*

Gastric Juice.—The discrepancies and contradictions of the accounts hitherto given of this secretion are in great degree due to the difficulties which its examination involves. The situation and office of the organ by which it is secreted, almost forbid its being obtained in a state of purity. Occasionally admixed with bile, always diluted with large quantities of saliva, as well as with alimentary substances themselves undergoing rapid metamorphosis, the gastric juice obtained from the digesting stomach is necessarily very impure. And in the fasting stomach, its quantity (especially in comparison with that of the residue of food and secretions then present) is too small for successful inquiry. The changes produced by disease, or consequent upon death, constitute further sources of error. It must be confessed that the most careful study of these circumstances scarcely reduces the discrepancies and contradictions above hinted at to any single consistent description. But it shows them to constitute conditions which, according as they are obviated, noticed, or neglected, respectively render any particular observations or experiments valid, comparable, or useless. And it forms the basis of those opinions on this controverted subject which are summed up in the following remarks.

Physical Properties.—Pure gastric juice is a transparent, limpid, structureless liquid, of a pale straw colour. Its taste is distinctly acid; and its smell a peculiar faint odour, characteristic of the species of animal from which it is derived, and allied to that of the blood. Its specific gravity is about 1003·3. In the healthy human adult the quantity secreted, during the twenty-four hours, probably ranges from ten to twenty pints; and under favourable circumstances,[1] its maximum in an hour may be estimated as not less than six or eight pints.

The *chemical properties* of the gastric juice are best noticed by successively considering its acid, saline, and animal constituents.

The *Gastric Acid.*—Omitting exceptional instances in which acetic, butyric, and other acids, allied to these products of organic decomposition, have been found in inefficient quantity in the contents of the stomach—and further eliminating the view taken by Blondlot, which ascribes the acidity of this secretion to the presence of the acid phosphate of lime—there are two views of sufficient importance to demand notice. One of these regards the gastric acid as the hydrochloric, and another as the lactic, acid. There can be no doubt that each of these acids has been repeatedly found in the gastric juice as the chief or sole cause of its acidity.[2] There is just as little doubt that they are sometimes present together, and that even in the same species and individual[3] the hydrochloric may be replaced by the lactic acid. While we shall see that either of the two would suffice to restore to a neutralized gastric juice its original digestive powers.

But a careful consideration of the facts hitherto known leads me to the opinion, that, in these latter cases, the lactic acid is always a secondary[1] and accidental product; and that the balance of evidence inclines decisively towards a single acid of the gastric juice, which, as normally secreted, owes its acidity exclusively to hydrochloric acid.[2]

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[1] Such as long fasting followed by a large meal of meat.

[2] Lactic acid by Chevreul, Lassaigne, Thomson, Lehmann, Payen, Bernard, Frerichs, and Smith; hydrochloric by Prout, Dunglison, Braconnot, Tiedemann, Enderlin, Schroeder, Bidder, and Schmidt.

[3] In Alexis St. Martin, as it would appear from a comparison of the analyses by Dunglison in 1833, and F. G. Smith in 1856.

Salts.—As regards the salts of the gastric juice, the details of an analysis of this secretion may be best comprehended (if not explained) by comparing them with a similar quantitative examination of the *liquor sanguinis.* The following table[3] exhibits such a comparison, for a thousand parts of both fluids.

	Liquor Sanguinis.	*Gastric Juice.*
Water	903·0	973·2
Animal matters	88·5	17·0
Mineral substances	8·6	9·8
Chlorine	3·6	5·6
Sodium	3·3	1·2
Potassium (in dog, ·2 ?)	·3	·6
Phosphoric acid	·2	·6
Phosphate of lime	·3	1·2
Phosphate of magnesia	·2	·2
(Lime corresponding to ·624 Ca. Cl.)		·3
	1000·0	1000·0

Hence, while most of the salts of the blood are present in increased quantity in the gastric juice, the chloride of sodium is so greatly diminished, as to lower the total saline contents of this secretion below those of the blood-liquor. While the amount of hydrochloric acid is so great, as not only to compensate this loss, but even to raise the total of its mineral constituents above that of the blood-liquor. The origin of this acid is obvious. Its mere quantity is sufficient to refer it to the chloride of sodium, which is the most plentiful chloride of the parent fluid. And the remarkable diminution in the sodium of the secreted fluid further confirms this view. Indeed, it is interesting to notice, that almost all the differences between the salts of the two fluids may be included in some such hypothesis as that of—(1) a rapid transudation of the blood-salts generally, followed by their concentration through an absorption of part of their water of solution; (2) a decomposition of about half of the chlorides, probably of the chloride of sodium;[1] (3) a return of the base of this salt into the blood. While it is evidently to a derivation of acid from some of the constituents of the latter fluid that we must refer the important fact established by Dr. Bence Jones,—namely, that, during digestion, the healthy urine loses the acidity proper to it at other periods.

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[1] [This opinion, which is also held by Professor Dunglison, of Philadelphia, has since been carried into conclusive details by him, as regards the earlier and later analyses of the gastric juice of Alexis St. Martin. In a private communication, Professor Dunglison has specially called my attention to the great dissimilarity of the two modes adopted for procuring the gastric juice, by himself in 1833, and by Dr. Smith in 1856. In the former case, it was obtained pure in the fasting state, by exciting the gastric mucous membrane with an elastic gum tube. In the latter, it could only be obtained after food had been taken; and hence it is not surprising that lactic acid was found. The secretion occupying the fasting stomach was not gastric juice, but a putrescent mucus.]

[2] Compare author's Essay "Stomach," p. 330.

[3] Here the composition of the gastric juice is calculated from an analysis by Schmidt; and that of the blood-liquor is quoted from Lehmann (Physiologische Chemie, Bd. ii., pp. 153, 179). To facilitate comparison, both analyses are reduced to one place of decimals; and the phosphate of lime of the former is divided into acid and neutral phosphate.

Organic substance, or Pepsine.—The addition of alcohol to pure gastric juice, or to a watery infusion of stomach, causes a white flocculent precipitate; which, when dried at a low temperature, forms a much less voluminous mass, of a yellowish grey colour, and a somewhat gummy appearance. This substance reddens litmus, and is soluble in cold water; but may be again precipitated from its aqueous solution by alcohol. Its ultimate composition closely resembles that of the various protein compounds, from which it differs chiefly in containing more nitrogen. And even its other chemical properties bear out this resemblance: its differences from many of the albuminous compounds consisting chiefly in the fact, that it is not precipitated from its watery solution by some of the salts which would throw down dissolved albumen. Allowing for variations due to impurities, the reactions due to gastric juice, and probably of pure pepsine, are as follows. It is not precipitated by heat, ferro-cyanide of potassium, sulphate of copper, alum, chloride of iron, or mineral acids. It is precipitated, though not completely, by bichloride of mercury. Carbonates of the alkalies precipitate its lime-salts. And the soluble salts of silver and lead throw down the chlorides of these metals. In all of these instances a portion of the pepsine is carried down with the precipitate. In the case of the salts of lead, the greater part of the pepsine is thus deposited, but may be almost completely recovered by washing.

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[1] Such a decomposition would obviously present many analogies to electrolysis. But though the acid and base are certainly unloosed and separated, the process itself cannot be definitely referred to this cause in the existing state of our knowledge. We may, however, notice that both the quantity and quality of the chloride of sodium would render it a more probable source of the acid by such electrolytic action than any other of the salts present in the blood-liquor.

Action of the Gastric Juice.—The addition of a few drops of diluted muriatic acid to a solution of the above precipitate in cold water, constitutes a liquid which possesses energetic solvent powers over ordinary animal food. Hence the organic substance itself has been termed *pepsine* (*πέψις*, *concoctio*),—a name to which there can be no objection, so long as its meaning is confined within proper limits, and is not extended to imply a single and definite organic compound, capable of digesting all the alimentary principles.

Temperature exercises an important influence on the gastric solvent. At the ordinary temperature of the atmosphere, the action of the gastric juice is scarcely perceptible, even when continued during many hours. Lower degrees of cold suspend its action still more completely. Heated to the temperature of the body, namely, to about 100° Fahrenheit, it acts very energetically. A further accession of temperature at first increases, but soon injures, and finally destroys, all its digestive powers. The precise point at which this change of effect occurs is not clearly known; but it is probably at or near 120°. The dried pepsine of the artificial digestive fluid will, however, sustain a temperature of 160° without damage. But at a heat above this, it becomes wholly inactive, and partially insoluble. And the pepsine of pure gastric juice is stated by Dunglison to be insoluble in hot water.

Alcohol, acids, and alkalies, when applied in excess, have also a destructive influence on the digestive power of pepsine.

In the case of acids, this injurious effect is much less marked. As might have been expected from the constant reaction of the gastric juice, an acid is essential to its digestive efficacy;—indeed, we might almost say, to its very existence. Even that incomplete loss of acid, which is necessarily involved in the precipitation of its pepsine, must be compensated by an artificial acidulation, before an aqueous solution of this substance regains its former powers. Here, however, as in the case of heat, it is necessary that certain limits should be observed. About half the quantity of hydrochloric acid present in the gastric juice forms a tolerably effective fluid. But the normal proportion (about 3 parts per 1000) may be increased to three or four per cent., not only with impunity, but even with advantage.

The nature of the acid seems a matter of indifference. Nitric, phosphoric, sulphuric, acetic, and lactic acid, have all been successfully made use of. And the range of amount already specified for hydrochloric acid might, *a priori*, prepare us for the fact, that the requisite quantities of these acids seem solely related to their more or less dilute state; and do not allow us to recognise any trace of an equivalent proportion.

Applied in still larger quantities, all of these acids first weaken, and then destroy, the digestive power of the solution of pepsine. The comparative amount of injury inflicted by equal quantities of the different acids appears to depend (like their solvent efficacy) chiefly on the degree of their concentration.

The essential aid given by the acid is well shown by the effect of neutralizing a natural or artificial gastric juice with an alkali. Under these circumstances, it not only loses all action upon albuminous substances, but, if mixed with them, shares their putrefaction. Left to itself, however, its powers are only suspended, being renewable by the addition of an acid. The addition of a larger quantity of alkali permanently destroys all its solvent powers, and is followed by its rapid putrefaction.

But though an acid is thus one of the essential elements of a digestive fluid, it must not be thought that any such agent can imitate the gastric secretion, even when associated with an infusion of salvia, mucus, intestine, bladder, or other animal product or tissue. The solution accomplished by such fluids is excessively slow, superficial, and imperfect; and affords—not the new compound produced by gastric juice from albuminous substances, but a weak and turbid solution, which readily yields its dissolved constituents to ordinary reagents. And even in the case of an acidulated infusion of intestine—where the results are necessarily affected by the diffusion of gastric juice over the whole digestive canal—the nature and amount of the process do not allow it to be any way comparable with that effected by an artificial gastric fluid.

The effect of the neutral salts on artificial digestion has scarcely been investigated with all the attention it merits. But it is probable that many of these inorganic substances assist solution, when present in small quantities, but oppose it when added in excess. This is especially the case with chloride of sodium, the ordinary condiment of mankind and of many animals.

The effect of alcohol is also regulated by its amount and concentration. Diluted, it seems to have no chemical action whatever. In larger quantities, as before remarked, it precipitates pepsine. And in still greater excess, it permanently destroys all its digestive energy.

In respect to the solvent properties of the gastric juice on the various protein-compounds, an exact determination of the quantity of pepsine which these substances require for their solution, would greatly assist us in solving many problems with respect to the chemistry of digestion. But the estimates derived from actual experiment are very conflicting—if, indeed, they can be considered really comparable. It may, however, be estimated, that one part of pepsine will dissolve about fifteen of moist and finely divided albumen: while the gastric juice itself possesses the power of dissolving from fifteen to twenty per cent. of its weight of the same substance.

The gastric juice dissolves, not only the various protein-compounds, but gelatin, chondrin, and gluten. In doing so, it arrests (and generally removes) all decomposition or putrefaction which they may be undergoing. The mere physical condition of the substances to be dissolved remarkably influences the rate of the process: density and bulk rendering it slow, while conversely, it is accelerated by minute division. The quantity of solvent required varies with the nature and aggregation of the particular substance.

In any case, the ultimate effect is that of a complete solution, containing a substance which (as shown by Mialhe and Lehmann), whatever the substance originally dissolved, possesses certain properties, entitling it to the name of *peptone*.

Peptone.—The following properties are common to all kinds of peptone. Reduced to the solid form by careful evaporation, it is a white or yellowish-white substance; almost tasteless and inodorous; very soluble in water; but insoluble in alcohol of eighty-three per cent. Its watery solution reddens litmus; and is precipitated by chlorine, tannic acid, and metallic salts; but is unaffected by boiling, by acids, or by alkalies. With alkalies and bases, it forms very soluble neutral compounds or salts. An aqueous solution of these is still less precipitable by reagents than one of peptone itself. Thus it is only thrown down by tannic acid, bichloride of mercury, and a mixture of the acetates of ammonia and lead;—the acetate of lead, and the ferrocyanide of potassium, causing but a faint cloudiness; and even concentrated acids, nitrate of silver, and alum, having no effect.

The ultimate chemical composition of any particular peptone so closely resembles that of the substance from which it is formed as scarcely to require any further notice.

In speaking of these chemical phenomena of stomach-digestion, there remains but to notice, that the addition of water, or a small quantity of fresh acid, is capable of restoring some of its original digestive powers to saturated gastric juice, or to a solution of peptone.

The above properties of the gastric juice naturally suggest the question—What is the nature of its action?

In answer to this question we may premise, that it is obviously no simple process of solution by a dilute acid; no mere contactive influence (like that of spongy platinum in the acetification of alcohol); no mere fermentation (like that excited by yeast in a solution of sugar); no mere complex acid combining with protein-compounds as bases (as in the "hydrochloro-pepsic" view propounded by Schmidt).

If we must connect the above details by some theory, we may first remark, that the gastric juice dissolves protein-compounds; that it renders them highly soluble; and that it assimilates their form and reactions to its own, without changing their composition. For any parallel to such a process, we can only look to those lower degrees of chemical action, where solution and combination, adhesion and affinity, may be supposed to meet and merge into each other; where proportions are tolerably definite, but true equivalents indistinct; and where, though form is changed and reactions modified, elementary composition remains little affected. Actions of such a kind may be found in the union of many substances with water, or its elements, to form the compounds called hydrates. And the conversion of protein into peptone, by the gastric juice, presents so many analogies to the formation of a hydrate,[1] that it would almost seem as if the chief office of this secretion were

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[1] The grounds of this view may be briefly summed up as follows. The great change of solubility undergone by albumen in its conversion by pepsine into peptone—or rather, the failure of various reagents to combine with and precipitate its mass—point to a change, of which it may certainly be said that it is just as distinct from mere solution in water, as it is from any real alteration in the proportions of the elements of the modified albumen; and that, unless we suppose the combination of pepsine with albumen to constitute the process itself, we are almost left (by exclusion) to the supposition of water being the modifying or combining substance. The strong attraction of pepsine for water confirms this view, as does also the fact that both pepsine and peptone bear heat when dry, though destroyed by it when moist; in other words, that it is only by the changes which heat impresses on their water of moisture, as distinct from any mere water of combination, that their efficiency is affected. It is further to be noted, that pepsine is, as it were, imitated by water; in so far as that the addition of water to a saturated solution of peptone gifts it with some of the energy of pepsine. And a variety of circumstances concur to render the analogy still more characteristic; and, indeed, exhibit pepsine as an agent only differing from peptone in the sense of its spontaneously undergoing, with disproportionate slowness, the same change which it transfers to albumen with extraordinary speed and completeness; and of which peptone is the final product in much the same sense as an artificial gastric juice kept too long loses all its powers, though perfectly free from the slightest taint of decomposition, and from any discernible change in appearance, reaction, or composition. Nay more, there are strong reasons for concluding that in at least the later stages of gastric digestion, the process of conversion is itself carried on by the product rather than by the original agent, by the peptone rather than by the gastric juice. For a digesting gastric juice seems to retain its power, and continue its action, long after a precisely similar juice, to which no albumen has been added, has lost all its powers.

Pepsine, then, seems to be an organic substance in course of hydration, and capable of propagating this process to albumen brought into contact with it. The hydration thus effected in albumen is itself capable of at least a partial propagation to fresh albumen. But, unlike the action of some ferments, the process is in so close a dependence on the whole organism, as to require not merely to be started, but to be reinforced, (and therefore to be quantitatively excited) by the acidulated pepsine or gastric juice. And while the quantity of dissolved albumen is thus in close relation to that of the secretion of the stomach, it is highly probable that this in its turn has an equally intimate dependence on nutrition generally: being derived that of enabling water to combine with the various members of the albuminous groups of alimentary substances; in order to their acquiring that solubility, and uniformity of constitution, which probably must precede their admission into the current of the blood. To this indication of a theory, I will only add, that the mode in which a definite quantity of the organic principle takes part in such a process cannot even be conjectured. Its action certainly appears no way comparable to the effect of diastase on starch, or of emulsine on amygdaline. It seems to be an assimilation, in the strictest chemical sense. It is not impossible that the acid commences the process by a slight, though genuine, solution of the more resisting substances. And at any rate, this constituent seems to have the power of checking putrefaction, if not of arresting all metamorphosis, in the other ingredients of the secretion; like the small quantity of oil of vitriol which is added by the chemist to hydrocyanic acid with the same object.

Process of Secretion.—The process by which the gastric juice is secreted from the mucous membrane of the stomach can scarcely be regarded as known, even in its larger phenomena. To correct some prevalent errors, and to offer a conjecture which future researches alone can fully substantiate, is all that the author would attempt here.

The secretion of gastric juice is not effected by any expulsion of the glandular contents of the stomach-tubes. Their elaborate

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from the tissues (even if proximately formed in the blood) in amounts which exactly express their changes, and the consequent need of their replacement.

In this "hydration," however, we seem to distinguish two stages, if not two elements, of which the latter only represents the chemical process ordinarily known by the term. In the first instance, water seems to combine with albumen physically, first swelling up its mass, and then rendering it transparent, soluble, invisible, but yet allowing it to be precipitated by most of its usual reagents. To this rapidly succeeds a more complete metamorphosis; in which the reactions of albumen are for the most part lost. The former stage suggests something quite unlike any ordinary process of solution; and it is quite specific to the gastric juice. The latter suggests a new grouping of the albuminous and aqueous elements. Both collectively make up a close parallel to the water of organization, which, tenaciously retained by all animal tissues, is not only the very condition of their existence, but is especially the agent of that changeableness the results of which we sum up by the term of Life, and the mediator of their physical properties. In these tissues too, as in pepsine and peptone, we may observe the reversal, as it were, of the stages of albuminous digestion in the result of heat, which first dries up, and then destroys, the bodily substance.

and dimorphous structure might alone suffice to prove this proposition. And the mere quantitative objections to such a view are scarcely less conclusive.[1] But it is better refuted by two facts : (1) that during every stage of gastric digestion, the tubes may be seen with precisely the same form, size, arrangement, and contents,[2] which they exhibit during the fasting state; and (2) that the pure gastric juice is completely structureless.

As regards the visible details of the act of secretion, Dr. Beaumont has reported some interesting observations. He made use of magnifying glasses, by the aid of which he could distinguish the spheroidal glandular follicles, and the papillæ situated in their interstices. These papillæ, or *villi*, he found to be scarcely visible until food was applied to the mucous membrane, when they underwent a kind of erection, and protruded from its surface in the shape of small sharp processes. (Compare Figs. 6, 10.) From these, according to this faithful observer, the gastric juice appears to exsude. Its secretion begins by the gradual appearance of innumerable lucid specks, which are smaller than the mucous follicles. These specks or points rise through the transparent mucous coat; and seeming to burst, discharge themselves upon the very points of these vascular papillæ, as a thin, transparent, colourless, limpid, acid fluid, which collects in small drops, trickles down their sides, and spreads over the whole gastric surface.[3]

It is evident that the above description closely corresponds with what would be seen on looking at the surface of the skin with a lens during the commencement of a violent sweat. For though, as a matter of anatomy, we know that there are no apertures of stomach-tubes on the ridges and processes which stud its free surface, yet, just as the structure of these tubes shews that it is from them the secretion comes, so, on physical grounds, the gastric juice would naturally tend to accumulate in drops on the projections of the mucous membrane, before spreading as a layer of liquid over the general cavitary surface of the stomach.

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[1] This expulsive view, applied to the human stomach during digestion, would imply the entire reconstruction of its cell-growth from 60 to 100 times in one hour!

[2] The greater softness and delicacy of these contents is no real exception to this fact.

[3] So thoroughly persuaded was Dr. Beaumont (*op. cit.*) that the fluid exsuded from the papillæ alone, that he had not the least doubt the excretory ducts of the follicles were inclosed in these villi, and terminated in the lucid specks just alluded to; although he admits that he could not see any apertures here.

In any further surmises as to the details of the secretory process in the stomach, it may be well to note (1) the fact discovered by Bernard, and repeatedly verified by myself, that it is only (or at any rate chiefly[1]) the surface of the mucous membrane which shews an acid reaction, either in the digesting or fasting state; and (2) the anatomical elements specially connected with this secretion.

As respects the first of these *data*, it points to the secretory process being only completed near the open ends of the tubes; and concurs with many other circumstances to suggest, for this process in the stomach, a view which is almost forced upon us in the case of other secreting organs. That, whatever be its exact details, the preparation of the gastric juice, begun in the blind ends of the tubes, is only completed at or near their opposite or open extremities; that, at any rate as regards the acid ingredient, the process is far more likely to be the subtraction, from a liquid allied to blood-liquor, of those materials which by their withdrawal would leave gastric juice, rather than any mere construction and effusion of this secretion; and that, in assuming such a selective or preferential absorption, we are only attributing to the stomach a special form and intensity of a process which we are forced to suppose operating in the kidney, as well as in the whole alimentary canal:—such are the physiological opinions which a careful study of the anatomy of these tissues would suggest.

To allot the two elements of the secretion—acid and pepsine—to two corresponding structures, is a much more doubtful and uncertain task. Nevertheless, the close resemblance of the axial epithelium of the true stomach-tube to the epithelium of the salivary and pancreatic glands, suggests that the somewhat analogous ptyaline and pepsine possess, in this minute epithelium, what is, anatomically, an analogous source. While the unprecedented and mysterious process which (as by an electrolysis) separates the chloride of sodium into acid and base, may be surmised to be effected by the equally characteristic and special structure—the large oval gastric cell. That the two co-extensive structures collectively achieve the secretion of both pepsine and acid, is conclusively shewn by the stomachs of innumerable animals of the Vertebrate class, in whom[1] I have found that the stomach, as ordinarily defined by the pyloric valve, includes a large extent of mucous membrane devoid of both these structures, and equally devoid (as demonstrated by careful experiments on artificial digestion) of the proper gastric function.

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[1] In verifying this observation, I have sometimes found, below the surface, a faint acidity, such as might be derived from the mere imbibition of the acid fluid from above.

It is hardly necessary to point out the admirable manner in which the structure and arrangements of the *quasi*-venous capillaries in and beneath the gastric ridges (p. 23) would facilitate their absorptive function. Their position, their size, the probable swiftness of their current, and the strong suction-force thus exercised, have an obvious relation to that vast and rapid withdrawal of peptone and grape-sugar from the gastric contents which they evidently effect.

A controversy of long standing as to the cause of that immunity evidently enjoyed by the stomach from the solvent action of its own secretion, has at length been closed by the experiments of Dr. Pavy. He has shown that it is neither to any abstract vitality of the living stomach, nor even to the continual dilution (and therefore neutralization) of its interstitial liquids by the salivary or other contents of the organ occupying its surface, that this immunity is to be referred; but to a protection afforded the stomach by the constant irrigation of the organ with circulating blood. His well-selected experiments prove that the mass of the organ is thus, as it were, perpetually neutralized by the alkaline blood-stream, and thus kept free from the process of solution for which a free acid is a necessary condition; and that while an arrest of the circulation permits the stomach to dissolve its own walls, this solvent effect is aided by artificial acidulation, and impeded by alkalizing, much in the degree which on such a theory might be predicated. The minor details by prosecution into which this theory will completely square with *all* that is as yet known of stomach-digestion are not quite complete. But they are perhaps (see p. 34) near at hand. Meanwhile, Dr. Pavy's discovery can hardly be rated too highly, as to both the healthy and diseased stomach, (see Lecture iii.).

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[1] "Medico-Chirurgical Review," July, 1861.

We may end these remarks by a glance at the share of the stomach in digestion generally.

The mastication and insalivation of the food is immediately followed by its *deglutition,* which propels the pulpy or semi-fluid mass it now forms into the stomach.

On entering this organ, it is subjected to a special act of *gastric digestion,* the total duration of which may be estimated as averaging two hours.

The energetic action of the mixed saliva is not affected by the gastric juice secreted by the stomach. Much of the starch of the food is probably converted into sugar during the short sojourn of the element in this cavity. The sugar thus produced would seem to be absorbed by the vessels of the gastric mucous membrane with extraordinary rapidity. The water, salts, and soluble organic compounds of the food are similarly taken up. And the gastric juice attacks and dissolves the proteinous element of the food. The perfection of this process of solution depends on the mechanical state of the substances concerned, and on the quantity and efficiency of the active liquid. Of the resulting solution or peptone, part is immediately absorbed by the gastric vessels, while part passes on into the duodenum, in company with protein which has not yielded to the solvent process, as well as with unsaturated gastric juice. Much of this protein ultimately becomes dissolved, and with the peptone which accompanies it, is taken up by the veins of the intestine. That, of all the secretions poured into the alimentary canal, the gastric juice alone has the power of converting the albuminous compounds into peptone—is a proposition which has too direct a bearing on the function and diseases of the stomach to be omitted here, though the reasons which, in my opinion, demand its provisional reception, cannot here be fully adduced. But while referring to them,[1] I may add, that

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[1] It is only with respect to the pancreatic juice that this proposition can be contested upon anything like plausible grounds.

Allowing for dilution and impurity, the infusion of secretory tissue is found to behave, in experiments on artificial digestion, like the secretion itself. Artificial gastric juice converts albumen into peptone, as does the natural secretion ; infusion of salivary gland transforms starch into sugar, as does saliva. And hence the utter it is evident the gastric juice retains its digestive efficacy after passing the pylorus; and that it is to the presence of this secretion (concentrated, perhaps, by absorption) that the solvent powers of this kind, attributed by some physiologists to the intestinal juice, are probably due.

failure of the infusion of intestine, whether neutral, acid, or alkaline, to dissolve albumen, or to transform starch, gives a decision against the possession of such power by the intestinal juice, which any equivocal evidence derivable from vivisections (especially when contradicted by more trustworthy lesions) quite fails to refute. Indeed, the necessary admixture of many secretions and substances in the intestinal canal of the living animal almost defies all satisfactory experiment.

In the case of the infusion of pancreas, however, a process of solution really does obtain: and it is the kind of solution, its postponement for many hours, and its apparently inseparable connexion with utter putrefaction, which militate against its constituting a function identical with that of the stomach in the living body. That albumen, when digested in pancreatic infusion, crumbles down, after many hours, into a kind of putrescent peptone, is a remarkable fact. But, limited to these propositions, the alleged digestive power of the pancreas on albumen seems to me reduced to little more than an elaboration of the statements long made by Bernard, as to the extraordinary proneness of pancreatic juice to decomposition. It is doubtless an important detail of the hitherto almost unknown chemistry of putrefaction. It suggests the discovery of relations between digestion and putrefaction, of surpassing interest and significance. It permits the surmise that, modified or held in check by other digestive agents, the pancreatic secretion may even in some way supplement or conclude, in the intestine, the gastric metamorphosis of albumen. But, in so far as it establishes long delay, and complete putrefaction, to be essential conditions of this process of solution, in so far does it deny the proposition that it is the office of the pancreas to dissolve and change albumen in the way in which the metamorphosis thus summed up is brought about by the stomach. (Compare the Author's Paper *On Digestion in the Vertebrate Series*, "Medico-Chirurgical Review," July, 1861.)

LECTURE I.

Introduction—Symptoms of Gastric Disease generally—Pain—Eructation, Regurgitation, Vomiting—Hæmorrhage—Flatulence.

The lectures I now commence are so few in number, and treat of so large and important a subject, that I can scarcely permit myself to occupy a minute in introducing them to your notice. But I may premise, that the reductions lately effected in the number of lectures entering into the medical curriculum are nowhere more seriously felt than in the Course of Physiology, where they especially limit that close and continual application of this science to the healing art, implied in the old name for this Course, "Institutes of Medicine." And it is with the purpose of supplying this deficiency, as respects the physiology of gastric digestion, as well as of putting before you the results of some special researches of my own, that I have obtained the sanction of the Hospital authorities for delivering this brief supplementary course of lectures on the Diseases of the Stomach.

I need say little to enhance my subject. The diseases of the stomach are not only some of the most frequent and curable which will hereafter claim your skill as practitioners, but they have a special importance from their bearing on all other maladies. No matter what the organ, or what the disease, it is by the stomach you will have both to assault the enemy, and to victual the beleaguered fortress of life you come to help. Indeed, the classical fable of '*The Belly and the Members*,' may suffice to indicate how the income and expenditure, the pleasures and pains, the health and disease, of the stomach, are alike vicarious and ministerial. The stomach, in short, represents in the constitution a kind of Chancellor of the Exchequer, who (if you like to pursue the analogy) has his budget dictated to him by the requirements of the various departments, and is not always the leader, even in the Lower House.

The interest thus attaching to these diseases is increased by their obscurity. For the physiology of the stomach is but now emerging from an uncertainty, such as has necessarily involved many of its pathological changes. These changes, again, are often accompanied by little evidence of their occurrence in the living body. Indeed, exact physical information concerning them is almost denied us. The aids to diagnosis afforded by auscultation in the diseases of the thoracic viscera, and by chemistry in those of the urinary apparatus, scarcely find any parallel in the maladies of an organ, which executes its work without perceptible sound or movement, and only dismisses its products from the body after a complex series of changes and admixtures. And although (as already stated) its disorders are both frequent and curable, and even its symptoms not difficult to recognise, still these very facts, which have doubtless favoured the acquisition of information, seem rather to have misled than facilitated stricter pathological inquiries; and have thus rendered our knowledge of the diseases of the stomach diffuse rather than profound, and useful rather than accurate.

To diminish this obscurity, and to add to the existing knowledge of these diseases, is one of the objects of the following lectures. And as I shall assume that the Physiological Course has already acquainted you with the propositions by which these lectures will be illustrated and sustained, so I may fairly ask you to apply here other branches of science you have learnt elsewhere. It is only by the diligent use of all our existing means of inquiry, and not by idly waiting for the discovery of new ones—only by putting our shoulders to the wheel, instead of merely calling upon Jupiter—that we can hope to advance in the study of these or any other maladies. Hence, while we shall look to physiology to explain their symptoms, to aid their prognosis, and to dictate their treatment—and shall find, in the occasional successes of modern quackery, the strongest confirmation of what physiology teaches as to the effect of proper food, air, and exercise on these maladies of the digestive apparatus—we shall not neglect other means of equal value. The careful physical examination of the belly will afford us information analogous to that yielded by percussion and auscultation in thoracic disease. The collection and comparison of the histories of large numbers of cases will teach us which are the more constant and essential of their symptoms and appearances; and the minute study of these symptoms, in contrast with the lesions found after death, will sometimes enable us to refine our diagnosis to a degree which appears to border on temerity. Lastly, with suitable precautions against the many sources of error connected with the digestive canal, the chemical and microscopical examination of the substances expelled from it will sometimes afford invaluable information respecting its state.

SYMPTOMS OF GASTRIC DISEASE GENERALLY.

To-day we consider the chief symptoms of gastric derangement, in such a general way as may prepare us hereafter to interpret their connexion with the several diseases of the stomach.

Pain.—Amongst such abnormal phenomena, pain is naturally the first to claim our notice. You are aware that the situation and office of the stomach render quite unnecessary its endowment with that accurate appreciation of the mechanical and thermical properties of objects, in virtue of which the skin forms so efficient a protection to the organs it envelopes; and that, in the stomach, all sensation of this kind is abnormal. Hence, if we except certain vague feelings but partially referable to this origin—the feeling of hunger periodically calling for food, and of comfort and satiety following its ingestion—we may safely subscribe to the popular *dictum*, that a healthy person ought not to be conscious of having a stomach at all. The substances he eats and drinks may range from a warmth of 120° to a cold of 32° Fahr.; and may differ scarcely less widely in their physical properties and arrangement. But when once they have traversed his œsophagus, and reached his stomach, these peculiarities are utterly beyond his recognition. In short, the stomach is devoid of all common sensibility.

But we are not therefore to imagine this important organ really insensible to stimulation; or to suppose that, because it is not every moment arousing the brain of its master, and demanding his forethought or exertion, it remains unimpressed and inactive. On the contrary, we must rather conclude that it has a special sensibility of its own; not one whit less marvellous (but surely more so) for possessing a certain independence of the cerebro-spinal centre. Closely related to this centre by the feelings of hunger and satiety—nay more, dictating to it (so to speak) those exertions which the proper alternation of these two states imperiously demands from the mass of mankind—it has a sphere of action altogether its own. And the study of digestion has shown us how admirably and silently the stomach fulfils its various and complex tasks; and how, incidentally to these, the unfelt particle of food no sooner touches its mucous surface, than it excites the flow of a variety of secretions, both far and near, and provokes movements in the muscular substance of its walls and vessels, as well as in the analogous structures of neighbouring parts. To these acts, which respectively constitute the sensation and motion of the healthy stomach, its morbid states afford an instructive parallel. And just as the kind of sensibility specific to a healthy muscle —the feeling of its strength, its equilibrium, its measurable force—seems to be traceable, by gradual modifications, through healthy fatigue to the feverish soreness of over-exertion, and through this to the universal muscular pain and prostration of various grave general ailments; so the indistinct sensation of the healthy stomach affords us the best clue to the acute sensibility of the diseased one; and allows us to trace a scale of similar kind—from satiety to repletion; from repletion to distension and weight in the epigastrium; and from hence to the dull, heavy aching of dyspepsia, the gnawing or burning pain of ulcer, and the sharp agony of cancer of the stomach.

How nearly the sensibility proper to the stomach approaches (and even implies) that appreciation of undue stimulus which constitutes its sensibility to pain, may be seen in the accurate observations of Dr. Beaumont on the living subject. In this well-known case, the introduction into St. Martin's stomach of the thermometer bulb, or of the tube by which Dr. Beaumont provoked and procured the gastric secretion, often gave rise to considerable pain and soreness;[1] at other times, to more general feelings, (vertigo, faintness, pallor, dimness of sight, sinking, &c.,) which seem to have been occasionally unaccompanied by any local sensation. These local feelings were exactly imitated by less mechanical causes, such as casual dyspepsia or intemperance.

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[1] These sensations do not seem to have been accompanied by anything equivalent

The various diseases of the stomach will hereafter acquaint us with the more characteristic varieties of gastric pain: at present, we need only notice a few points connected with its nature and origin.

And firstly, as regards its *locality,* in numerous cases pain referred to distant organs really originates in the stomach. In such cases, however, we must often doubt whether that state of the stomach which evidently conditionates the pain—suppose a headache or a toothache—does so by a direct or indirect causation. Thus, on the one hand, the gastric irritation may act by exciting, in some intervening nerve and centre, a condition such as is either referred from this centre to that periphery of another nerve, which is the seat of the pain, or really brings about local changes here. On the other hand, the distant part, already the seat of some change, may only be excited to pain by an additional irritation, which is either transmitted to it from the stomach, or acts by its mere depressing influence on the centre of this distant part. In either case, the phenomenon is so complex, and is producible by the derangements of so many other organs, that, while allowing the frequency and importance of its connexion with the stomach, we are hardly entitled to discuss it as gastric pain.

But, in a large proportion of cases, pain originating in the stomach is referred to the region occupied by this organ; and with sufficient accuracy, to render the precise situation of the pain a matter deserving of notice.

Great or specific exactness of such local reference of course fails us. From the physiology of pain itself, it would be easy

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to spasm of the pylorus, much as the energetic contraction proper to the whole pyloric sac probably aided to bring about the physical conditions by which the pain was provoked. The notion of a cramp of the stomach analogous to a cramp of the leg (and still more of a cramp of the pylorus produced by morsels of undigested food), is, indeed, a mere assumption;—the sudden and violent pain which is all such a term really implies, being certainly producible by a variety of causes, and doubtfully ever due to that cause which it claims to connote.

to deduce why, as experience shows, a certain indistinctness generally belongs to this abnormal sensation—is, indeed, a rule, of which pains produced by lesions involving the skin or other analogous organs, are the easily explained exceptions. The accurate perception of objects demands, amongst other things, a specific organization at the periphery of the nerves by which it is mediated. Hence the abnormal stimulus or injury which can bring about pain, whether it is applied to the trunk or the branches of a sensitive nerve, is only appreciated with anything like local exactness when it is applied to its terminal distribution.[1] And the nervous arrangements of the stomach are calculated still further to increase this uncertainty and indistinctness: inasmuch as they only connect the organ with the cerebral centre of all perception by collateral communications, and through intervening ganglia or independent centres, such as render the mere transmission of the stimulus far less certain, and its modification during this transit far more probable than would be the case in a stimulated cerebro-spinal nerve. The pain of gastric derangement does, indeed, possess all those attributes which the anatomy of the gastric nerves in Man, and the study of their arrangement and office in various Mammalia, would lead us to expect. It is often absent when we might fairly expect it to be present; present when we have no clue to any local cause for it; variable or intermittent without any assignable reason; and lastly, even when there is a definite and circumscribed lesion, gives but a wide and indistinct indication of its place, or perhaps refers it to a point of the abdominal surface much above or below, to the right or to the left of, its true situation.

This indistinctness may, however, be partially ascribed to other causes:—namely, to the situation of the stomach; to the number of organs with which it is in contact; and to the frequent displacements which their movements and its own permit, and even necessitate. Thus, in pointing to the pit of the stomach as the seat of his pain, the patient, however exact in his sensations, can rarely indicate one of the three directions of space—the depth to which his sensation of pain is to be referred. The various changes of position associated with the movements of the heart, the lungs, the diaphragm, the liver, the intestines, and the stomach—can also rapidly modify the exact situation of the latter organ, and thus increase the area of indistinctness. Besides, in pointing to the epigastrium, the patient is pointing to a kind of focus, formed by the convergence and attachment of a number of important organs, and hence liable to be occupied by the pain which the lesion of any one of them can produce. Pericarditis, pleurisy, gall-stones, hepatic abscess, diaphragmatic lesions, emphysema of the lungs, and a variety of intestinal causes (amongst which I have myself verified an obstruction of the small intestine occupying the right iliac fossa)—any one of these can produce what is, strictly speaking, pain in the epigastrium, and therefore so far simulative of gastric pain. And though it is probable that the contrast of the latter with any one of the former would show a considerable difference—so much so, that few persons unfortunate enough to have experienced the two would be incapable of distinguishing them—yet ordinary language affords little means of expressing the differences thus felt, to say nothing of the rarity of such a direct comparison.

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[1] Perhaps the only glimpses of accuracy of sensation on the part of the stomach are (as we should expect) those in which distension distributes the morbid stimulus over a large surface of the organ. The "stoppage" of food referred to the cardia and pylorus respectively in disease of these apertures, as well as the similar sensation sometimes produced by an unnatural constriction in other parts of the stomach, well illustrate this rule. Like the curiously exact feeling of extreme repletion which has been noticed in general inflammatory thickening of the whole organ, even when attended with great contraction—this sensation, however accurate, deviates little from the ordinary rule of gastric sensibility above alluded to.

Without anticipating what must hereafter be said respecting the situations of pain in different gastric ailments, I would suggest (not without admitting their many exceptions) the following general rules as to its significance. The pain of gastric disease is grave in proportion not only to its severity, but also to its concentration and fixedness. In other words, a severe and continuous pain confined to a single spot of small size, is a more serious indication than one which, at times of equal (or nearly equal) severity, fluctuates in its different attacks, and ranges the epigastrium, of which it habitually occupies a wide area. Pain is graver in or near the median line; not only because (for many reasons which will readily suggest themselves) it is, *cæteris paribus*, more certainly gastric here than elsewhere, but because this situation (at least such is my opinion) indicates a more serious derangement of the innervation of the organ, than where the pain has a less exact correspondence with the solar plexus. Lastly, of all situations, a median and dorsal one (in anatomical language rachidian, and ranging from interscapular to lumbar), which is usually an addition and complication to a previous epigastric pain, is the most serious; so much so, that it will rarely be found associated with any but the graver gastric dyspepsias; and belongs chiefly to deep ulcers, or to cancerous lesions of the stomach, involving all its coats.

In all cases it is essential to add the information obtainable by pressure to the mere sensations complained of by the patient. It is rarely that severe or continuous pain affects the stomach without being associated with some soreness or tenderness to pressure. But when pain is sudden and temporary, and is attended with much flatulence, it is often relieved by gentle pressure. Hence the occurrence of either modification in any given case is capable of greatly influencing our opinion as to the nature of the pain. For, aided by a strict physical examination of the existing state of the stomach in respect of size and situation, it sometimes definitely refers to this organ a pain which, in its absence, could only be conjectured as gastric. In many cases, too, tenderness does far more. According as it is moderate or excessive, superficial or deep, localized or diffuse—features the due recognition of which often imply great delicacy and skill—it justifies a conclusion, not only as to the nature of the gastric malady, but even as to its seat: distinguishes, for example, general inflammation from ulceration or dyspepsia, or a lesion of the peritoneal from one of the mucous coat.

As respects the immediate causes of gastric pain, it is probably in most cases to be referred to undue distension of the vessels, irritating (one might almost conjecture stretching or otherwise injuring) the nerves distributed upon and within their coats. The great changes of calibre which the arteries and veins of the stomach so often undergo, in the sudden transitions of this organ from rest to activity, and *vice versâ*, are doubtless mediated by nerves, which supply their muscular coats, and have so far a motor function. Whether these vessels also possess correlative nerves, which have the distinct office of appreciating these and other changes, and are therefore (*pro tanto*) sensitive nerves, remains at present unknown. But on the whole, the violent gastric pains which sometimes occur quite independently of any perceptible lesion, and even (if we may trust the microscope) of any interstitial exsudation, are best explained as provoked by a physical irritation or injury of nerves —perhaps sometimes a mere excess of their natural stimulus— impinging rather upon the nerves of sub-mucous situation, distributed to the small arteries and veins of the stomach, than upon any ultimate nervous ramifications in the secretory structures themselves. (Compare pp. 23, 26.)

Amongst less immediate causes of pain, we may enumerate the following as the most obvious and important. Undue distension of the stomach, illustrated by some forms of pyloric obstruction, as well as (though more questionably)[1] by flatulence; local injury, eroding branches of nerves, and irritating their severed extremities, as in gastric ulceration; and the more variable (but probably scarcely less local) injuries producible by cancerous deposits. Each of these will require allusion in its respective place.

Vomiting, the next symptom of gastric derangement, is an act so closely connected with the healthy organization of the stomach (indeed so definite a constituent of the digestive process in many Vertebrata), that our cursory view of the physiology of the stomach would have been incomplete without such a brief description of its nature and mechanism as may be usefully recalled here.

Normally, the human stomach propels its contents onwards into the duodenum. But, under various abnormal circumstances, this direction is reversed, so that the gastric contents take a backward course through the œsophagus, re-enter the mouth, and are thence expelled the body. Such a reversal obtains in the acts of eructation, regurgitation, and vomiting. And these three acts, whatever their difference of detail, further agree in the conditions of their occurrence, in so far that they all require (1,) an open cardia; (2,) a closed pylorus; and (3,) a com-

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[1] Compare the remarks on flatulence at the end of this lecture.

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pressed stomach;—compressed, that is, either by its own muscular contractions, or by some extrinsic pressure.

In *eructation* or belching, part of the gaseous content of the stomach is expelled from the mouth. How the cardia becomes patulous, it is not easy to decide. The stomach so frequently contains gases, and (as may be well seen in vivisections) the cardia resists their expulsion so efficiently, that we can scarcely suppose this aperture would be opened by the mere evolution of any quantity of aeriform fluid in the stomach; or would allow of any considerable leakage, save by a definite relaxation of its walls. The agents of the expulsive act are also somewhat doubtful. For though the act appears to coincide with an expiratory effort, and sometimes with even a closure of the glottis, yet the abdominal pressure thus brought to bear on the stomach is certainly by no means violent. But such mobile fluids would scarcely require any remarkable effort for their expulsion. Indeed, those contractions of the stomach which are necessarily present when the act of eructation occurs during gastric digestion, appear quite sufficient to commence, if not to complete, the expulsion of gases from the organ.[1]

In *regurgitation*, some of the liquid contents of the stomach are returned into the mouth. This act seems to be often a mere accidental complication of the preceding, a small quantity of liquid being carried up the œsophagus together with an eructation of gaseous fluid. In other instances, however, the liquid arises alone; and so quietly, that it is only perceived when it reaches the fauces and back of the tongue, where its taste causes it to be recognised. It is probable that the nature of this expulsion is closely akin to that of eructation: the abdominal pressure playing, if anything, a still more subordinate part.

The act of *vomiting* differs from the two preceding, not only in the quantity and quality of the matters (solid as well as liquid) which it expels from the stomach, and in the far greater energy and completeness of the expulsive efforts, but also in the fact—that a violent pressure, extrinsic to the organ, is the chief agent of the process.

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[1] In voluntary eructation, a kind of twitch, quite unlike ordinary deglutition, introduces air from the pharynx into the upper end of the œsophagus, whence it appears to be propelled into the stomach. Soon after it reaches this organ, it seems to be returned through the still patulous cardia by a well-marked expiratory or abdominal pressure; during which the glottis appears to be at least partially closed. And as the air thus artificially introduced into the stomach is often accompanied in its expulsion by part of the gases previously contained in the organ, the voluntary and involuntary forms of eructation almost merge into each other.

As respects this abdominal pressure, it will be remembered that, in ordinary respiration, the viscera of the belly sustain but a moderate compression. For during inspiration, the contraction and descent of the diaphragm exactly coincide with a relaxation of the muscular walls of the abdomen; while, during expiration, the compression which these exercise is neutralized by the recession or ascent of the now relaxed diaphragm into the thorax. Hence the movable contents of the belly escape all violent pressure; and merely transfer, as it were, a slight force from the upper to the anterior wall of this cavity, and conversely. But if, while the diaphragm remains depressed and contracted, the abdominal muscles also vigorously contract, the whole force of either of these two muscular strata may be regarded as compressing the viscera within the abdominal cavity. And since many of these viscera are hollow organs, enclosing movable contents, and communicating with the exterior of the body, such a forcible pressure must expel their contents, so soon as their terminal orifices are thrown open—whether by relaxing, or by yielding to a superior force. In this way the abdominal pressure plays an important part, not only in vomiting, but also in defæcation, micturition, and parturition;—affording a powerful, though intermittent, force, in aid of those more continuous expulsive contractions which are effected by the muscular walls of the rectum, the bladder, and the uterus respectively.

There is ample evidence that the act of vomiting is effected mainly by this abdominal pressure, which is not only indispensable to it, but (as proved by vivisections) suffices to effect it when reduced to a contraction of the diaphragm, or of the abdominal muscles, or even to a slight muscular compression of the hypochondria.

The exact aid given by the contraction of the stomach is less capable of determination. That any such assistance not only can be, but often is, altogether dispensed with, it is scarcely possible to doubt. But, on the other hand, it seems equally certain that the abdominal pressure, to which the act of vomiting is chiefly attributable, is often accompanied, and assisted, by a contraction of the muscular wall of the stomach itself. And as might be expected, observation on Man and animals during life shows that this contraction specially engages, not only the pyloric valve, but the neighbouring muscular pyloric extremity of the organ; in movements which are probably rhythmic (contraction regularly alternating with relaxation), and peristaltic,[1] and which there is certainly no sufficient ground for supposing to be ever anti-peristaltic.

The phenomena of the act itself quite confirm the conclusions, as to its strictly co-ordinate nature and arrangement, arrived at by the physiological inquiry just summed up. Feelings of uneasiness, pain, or distension in the gastric region, generally precede an increased flow of saliva, and a loathing for food, which soon heightens into downright nausea. To these gastric symptoms accede others of a cerebral import: giddiness, languor, dimness of sight, or even headache. Next occur retchings, which seem to open the œsophagus and cardia, but not to be attended with any abdominal pressure; and which often have the effect of gradually distending the stomach with air, so as to facilitate the subsequent occurrence of vomiting. Finally, an uncontrollable impulse completely changes the action of the muscles of respiration, and brings into one moment two contractions, which usually alternate with each other. An energetic closure of the glottis follows the descent of the diaphragm, so that this muscular septum is fixed by the distension of the thorax, as well as by the contraction of its own fibres. At the same time, the abdominal muscles contract violently upon the stomach; and exercise a compression which not only expels its contents, but which, aided and increased by the contraction of various other muscles of the trunk, causes the head to become greatly congested. Hence the features become red and swollen; and the large veins of the face, temples, and neck, are visibly distended. The expulsion of the gastric contents is sometimes attended by considerable pain; which appears referable to the lower end of the œsophagus, and is probably due to the spasmodic compression of this tube by the diaphragm.[1] The discharge of the matters vomited through the pharynx and mouth appears to be aided by a kind of reversal of the movements of ordinary deglutition. But if the act be violent, and the quantity of liquid large, these movements lack their ordinary precision; so that a part of the matters expelled generally eludes the curtain of the soft palate, and traverses the respiratory channel of the nasal fossæ, to gush out of the nostrils. The subsequent phenomena vary with the origin of the vomiting;—where, as is often the case, it has a local cause, which is removed from the stomach by the act itself, the patient speedily recovers his normal condition.

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[1] Assuming the analogy of this contraction to that verified by Dr. Beaumont in the human subject, and by myself in the Dog, during the most active stage of gastric digestion—the rate of peristalsis, and the alternation of contraction with relaxation, would not be at all incompatible with a constriction of the whole pyloric sac lasting at least a minute: a duration which would more than cover the time actually occupied by any single expulsive act during the ordinary fit of vomiting. Compare the "Cyclopædia of Anatomy," article Stomach (Suppl. p. 313).

As regards the *causes* of vomiting, it is obvious that so complex and co-ordinate an act of various organs can only be accomplished through the intervention of that cerebro-spinal centre from which the nerves of these organs radiate. And hence we may distinguish these causes into two classes;—one in which the irritation provoking the act originates at the nervous centre; and one in which, arising at the periphery, it is transmitted thence to the centre, to be reflected into the various organs which effect the expulsive process. The first class is illustrated by the vomiting of cerebral injury or disease, as well as by the vomiting sometimes resulting from mental emotion: the second, not only by that ordinary form of vomiting which is excited by a direct irritation of the stomach itself, but by the numerous cases in which it follows an irritation of kindred or even dissimilar textures—such as irritation of the fauces, intestines, or peritoneum; disgusting sights, sounds, or smells; prolonged immersion in cold water; and even wounds of the extremities.

But even in the second of these classes the act of vomiting can only be regarded as "reflex" with two important qualifi-

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[1] As is shown by the fact, that a similar pain sometimes may be produced, independently of vomiting, by a voluntary abdominal pressure with a closed glottis.

cations. Firstly, there are good grounds for believing that a large number of emetic substances are not only quite as active when injected directly into the blood as when merely introduced into the stomach, but that, as shown by Magendie's celebrated experiment (in which tartar-emetic injected into the blood of a dog caused copious vomiting from a bladder which had been substituted for the stomach), whatever their local action on the stomach in the latter of these two cases, it is to a direct influence on the nervous centre that the act of vomiting must chiefly be referred. And secondly, though, in those numerous cases where the exciting cause is a mechanical irritation, the phenomena of vomiting sufficiently indicate the medulla oblongata to be that segment of the cerebro-spinal centre in which reflection towards the periphery occurs, still the ensuing movement is by no means a simple reflex act. On the contrary, it is effected by the concurrent action of so many organs, and with such characteristic and definite changes in their ordinary times, modes, and degrees of activity—the whole process is so complex, and so truly co-ordinate—that, far from limiting our attention to the reflected course which its exciting cause sometimes takes, we ought rather to regard this as quite a subordinate feature, and the act of vomiting as an involuntary or physical nervous action of the highest order.

Such a view of the nature of vomiting receives a remarkable illustration from an experiment of mine on the action of tartar-emetic,[1] which conclusively shows what we may almost specify as an object, a purpose, or at any rate a function, of this complex process. On injecting a solution of tartar-emetic into the superficial femoral vein of a dog, the mineral was found ten minutes after in the fluid contents of the stomach, and in a state of concentration much exceeding that in which it must have been mingled with the mass of the blood. In this instance, too, the poison had produced extreme prostration, but no vomiting. Taken in conjunction with Magendie's experiment already alluded to, it proves that the poison may first be secreted by the stomach into its cavity, and then removed from this cavity, by two processes which are essentially independent of each other, and of any local effect of the secreted drug, however aided by it. A similar process of secretion probably obtains in the case of the salts of other metals, as well as in that of various organic poisons:—the concurrence of such a process with the act of vomiting thus rendering the latter an element of the *vis medicatrix Naturæ*—an act by which the organism throws out a deadly poison like antimony, perhaps even a dangerous disease like fever.[1]

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[1] For the results of this experiment, made in the year 1850, see the article Stomach, "Cyclopædia of Anatomy;" and the "Lancet" for 1853, vol. ii. p. 599.

In some instances, the process of vomiting returns different portions of the gastric contents at different periods of time; so that, for example, the expulsion of a comparatively homogeneous fluid is followed, after a considerable interval, by that of crude masses of undigested food. Some of these cases perhaps depend on a peculiar hour-glass shape of the organ, or on a similar (but more temporary) shape produced by the muscular contraction of its middle, isolating part of the contents for a variable period. But most of them are probably referable to the weight, bulk, and situation of the substances contained in the stomach, and to the other mechanical circumstances which regulate the act of vomiting itself. The last efforts of a prolonged vomiting often bring up bile. But though there is nothing abnormal in the mere presence of this secretion in the fasting stomach, still the above circumstances show that its being vomited in comparative purity is generally an indication of the completeness or duration of the expulsive process. It is probably during the intervals of energetic vomiting, that it finds its way through the pylorus into the stomach. It may, however, be doubted whether the pylorus is always strictly occluded at the exact moment of the expulsive act; especially in those cases in which the intestines are themselves distended with fluid, such as must necessarily be exposed to the same pressure as the contents of the gastric cavity.

In alluding to vomiting as a symptom of gastric disturbance, we must begin by conceding, that cases sometimes occur, in which the vital powers are rapidly exhausted by violent and frequent vomiting, that defies all our efforts to relieve or arrest it; and in which our complete uncertainty as to the cause of this vomiting during the life of the patient is little affected by a necropsy, showing no apparent lesion in the stomach, or even (it is said) in any organ of the body. We cannot therefore augur the gastric origin of vomiting from its mere severity and frequency: nay, more, we must not expect that any single peculiarity of this symptom—such as its excitement by food, its association with epigastric pain, or even its expelling blood—will often suffice to decide its reference to the stomach.

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[1] Compare the author's "Essay on the Treatment of Continued Fever," the "Lancet," *loc. cit.*

One general rule respecting it we may, however, lay down. While it is to the aggregate of symptoms that we have to look for our diagnosis of the cause of vomiting in any given case, it may, I think, be propounded, that the facility with which an irritation produces vomiting varies (other things being equal) with the closeness of alliance between the stomach and the irritated part. For example, vomiting is excited more frequently and readily by an irritation of the duodenum or pharynx, than by an irritation of the jejunum or mouth respectively: and again, by irritation of the small intestine rather than of the large; of the mucous rather than of the peritoneal coat, throughout the whole canal; of the brain rather than of the integuments. Conversely (as may be noticed in pleurisy, pericarditis, aneurisms, and various endocardial lesions) vomiting is a grave symptom in many thoracic diseases, because (still *cæteris paribus* only) it implies a more serious mischief than would suffice to produce it in lesions of the abdominal cavity.

In the present state of our knowledge, we may usefully group these facts by the conjecture that, whatever the kind of cerebral disturbance necessary for the production of vomiting, a certain degree of irritation of afferent branches of the sympathetic system will generally suffice to excite it; and with a facility which appears to be, on the whole, tolerably proportionate to the closeness with which these branches are related to that great pre-vertebral centre of the abdominal sympathetic, formed by the semilunar ganglia and the solar plexus.

Among the vomitings produced by gastric derangements, we may distinguish the following varieties. Firstly, the vomiting brought about by sheer destruction of tissue, involving an abnormal irritation of the nerves laid bare at the seat of lesion:—a variety exemplified in simple and malignant ulceration, in wounds of the stomach, in corrosive poisoning; and characterized (as might be expected) by a remarkable amenability to the physical or chemical properties of substances brought into contact with the injured nerves (as in the ingestion of food). Secondly, the vomiting of obstruction, which is referable, not so much to the mere obstruction, as to the distension and violent muscular movement which is gradually brought about behind the occluded part; and which therefore varies, not only with the strictness of the occlusion, but with its proximity to the pylorus, its superficial extent, its disposition relatively to the muscular coat, and other circumstances of this kind. This variety of vomiting is often seen in cancer, and (a still better example) in cicatrized ulcer of the stomach. Thirdly, a kind of vomiting in which the gastric distension present appears mainly referable to a loss of contractile power by the muscular coat of the stomach (the structure of the organ remaining unchanged); and in which we must often doubt whether this failure of contractility is not caused by some nervous lesion itself answerable for the vomiting—whether, in short, the distension of the stomach is simply concurrent, or really causative in the process.[1]

Such allusions to the nature of vomiting may end by the general statement that, just as it is the kind of vomiting which dictates its treatment—so that, for example, the cerebral vomiting of sea-sickness is often relieved by food and stimulants, the vomiting of phthisis by whatever will diminish or mitigate cough—so the vomiting produced by gastric derangement is most accessible through the function of the organ: in other words, is most amenable to a treatment regulating the quantity and quality of the food, and the frequency of its ingestion. The great (though subordinate) usefulness of various drugs will be more fitly discussed hereafter.

Hæmorrhage.—In alluding to hæmorrhage in connexion with diseases of the stomach, it must be remembered that even in the gastric maladies to which it is incident, it only gives rise to what are, strictly speaking, *symptoms,* in those cases in which its occurrence is betrayed of matters expelled from the alimentary canal by stool or vomit. And hence, far from regarding *hæmatemesis* as the exclusive (or even the chief) indication in hæmorrhage in gastric disease, we must bear in mind, that blood effused into the stomach frequently passes off in the fæces without being vomited at all. While, considering that it is rarely or never vomited so completely and instantaneously as to prevent the passage of any portion of it into the intestine, it is obvious that this exit *per anum* is on the whole much the more frequent symptom of the two.[1]

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[1] Compare p. 67.

The two-fold uncertainty which attaches to most symptoms of disease is especially marked in the expulsion of blood as a symptom of gastric mischief. On the one hand, as above hinted, a slight and intermittent hæmorrhage into the stomach so often depends on casual circumstances for its detection, that we can never presume its non-occurrence because its results have not been observed. Nothing, indeed, but a constant and sedulous observation of the gastric and intestinal evacuations affords any valid conclusion of this kind. On the other hand, it is scarcely necessary to say, that a mere hæmorrhage into the intestinal canal by no means proves the stomach to be the locality of the effusion, still less a malady of this organ to be the cause of it; but leaves open a wide question as to both its source and cause—a question such as can only be decided by that process of induction which (consciously or otherwise) guides every scientific physician to a diagnosis.

Merely to enumerate the causes which may introduce blood into the digestive tube would be out of the limits of our subject. But the occasional results of epistaxis, hæmoptysis, gall-stones, cysts of the liver, and lesions of the pancreas, illustrate the obvious law:—that any lesion which brings about a hæmorrhage into the various cavities which communicate with the digestive tube, may indirectly give rise to the influx of blood into the stomach and bowels, and therefore to its expulsion thence.

But the causes of even a gastric hæmorrhage are sometimes remote from the organ, and independent of it. For

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[1] Hence the use of the term *hæmatemesis* requires that of some such cognate (and equally uncouth) term as *hæmatokopræsis*. And the term *melæna* bequeathed us by Hippocrates—a term which now confounds vomiting and purging, and connotes such a colour of the matters expelled as is neither constant nor essential—ought to be expunged from our nosology.

example, any mechanical obstruction to the portal system may bring about so violent a distension of the veins which form its commencement in the walls of the stomach and intestine, as finally to extravasate a variable quantity of their contents into either of these segments of the digestive tube. In this way cirrhosis of the liver, or tumours or deposits compressing the portal vein, may give rise to a hæmatemesis (more frequently, however, to a hæmorrhage *per anum*), which is really unconnected with any disease of the stomach itself, however it may embarrass, or even suspend, the functions of this organ.

In noticing the causes of gastric hæmorrhage, little need be said as to the old doctrine of hæmorrhage "by exhalation" through the coats of the vessels, apart from any solution of their continuity. For we now know that the walls of even the finest capillaries have no pores of appreciable magnitude, such as would be necessary for the transit of blood-corpuscles; and hence that the extravasation of these structures is proof positive of some vessel having been ruptured. That, amongst the myriads of these minute tubes present, the eye often fails to detect the exact vessel or vessels involved in the lesion, is of course not very surprising.

The anatomy of the vessels of the stomach is intimately connected with the phenomena of these hæmorrhages; especially as explaining the great facility and small risk of scanty extravasations, and the rarity and danger of larger ones. Thus in the kidney, hæmorrhage is comparatively rare and dangerous; and when it once occurs, is rendered extremely liable to return, or even to become continuous or chronic, by those very derangements of the renal circulation which the effusion itself tends to bring about. In the lungs, with perhaps a greater inherent liability to hæmorrhage, the physical and mechanical conditions of the respiratory act add an alarming element of danger; so that, for example, we may almost compare any casual lesion or injury of the pulmonary capillaries to a fissure, through which each inspiration tends to pump out the contents of the pulmonary artery. In the stomach, on the contrary, hæmorrhage often occurs under circumstances which imply little danger to life, sometimes even little derangement of health; may be frequent, and yet extremely scanty; and lastly, may be recovered from with such completeness, as to defy the most careful necropsy thereafter to distinguish its site, greatly as the arrangement of the vessels concerned sometimes facilitates their inspection.

The capillaries of the stomach, it will be remembered, are of two kinds: deep, inter-tubular (*t*, Fig. 9, p. 22), or gastric proper, as we may call them; and superficial (*r*, Fig. 9), or supra-tubular, which adjoin the general cavity of the organ. In rare instances, the former seem to constitute the chief or exclusive source of hæmorrhage, so as to fill more or less of the blind end of a stomach-tube with blood. But hæmorrhage is far more commonly confined to the latter—to those complex rings of capillaries (Fig. 10, p. 23) which surround the open mouths of the stomach-tubes, and directly underlie the ridges and papillæ intervening between their orifices. These vessels, extremely numerous, and possessing a size and arrangement such as renders them almost intermediate between true capillaries and veins, seem to be peculiarly exposed to the ordinary causes of extravasation. They evidently have all that liability to congestion which belongs to the commencement of the venous part of the circulation throughout the body. They have a situation which requires them to bear the brunt of the various agents introduced into the human stomach in the food. And their delicate membranous wall is only shielded from the physical and chemical action of these agents by a layer of columnar epithelium; the removal of which, though doubtless strictly abnormal, must at any rate be a frequent accident in the history of the digestive process. Indeed, if we think of the incidents of a single meal—the fluids and solids; the animal and vegetable substances; the peppers, acids, wines, liqueurs, spirits; the frozen and scalding, the hard and soft, matters which are sometimes brought into rapid and successive contact with the gastric mucous membrane (and this, too, at a time when the process of digestion itself implies a state somewhat akin to congestion, in so far as it distends and thins these delicate vascular and epithelial structures)—we can easily understand that (as Dr. Beaumont's observations[1] indicate) the soft columnar cells may be here and there stripped off, and the subjacent vessels here and there distended and ruptured, by a casual debauch or dyspepsia, to be restored by a few days of abstinence or temperance to their pristine perfection.

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[1] *Op. cit;* in which compare pp. 196, 250, 251, 255, 265.

Little, therefore, can be concluded from the detection of a small quantity of blood in the matters rejected from the stomach of a patient. However suspicious a circumstance, it is only by careful collation with other symptoms that it materially influences diagnosis. It proves a solution of continuity in the vessels yielding it; but leaves us to determine whether this rupture is due to vascular obstruction, to congestion, to desquamation, or to ulceration.

In larger quantity, gastric hæmorrhage has not only more gravity, but a more definite import. Thus, sometimes it presents sufficient of the characters of arterial or venous blood, to justify its reference to a vessel of one or other of these two kinds. And even more frequently it shows, by the clots it contains, that it must have been effused rapidly; either from one or two large, or from numerous small vessels. In other cases, it presents the peculiar black colour and tarry consistence which blood assumes by exposure to the digestive juices. Since this change of colour requires a certain time, as well as a certain amount of these juices, to produce, its occurrence in vomited blood generally indicates that the hæmorrhage has either been slowly effused, or is of but moderate amount. In blood discharged from the stomach by the bowels, of course no such rule will obtain; indeed, such a hæmorrhage will rarely fail to exhibit somewhat of this tarry colour and consistence, unless its quantity have been excessive, or its transit through the intestinal canal unusually rapid. The detection of hæmorrhage is sometimes rendered difficult by the admixture of blood with other substances of similar appearance: especially with various articles of food, and with more or less altered bile, occasionally even with morbid products. A careful examination[1] will, however, generally clear up any obscurity of this kind. Indeed, a mere dilution of the inspected matters with water usually suffices; or, if not, a microscopic examination rarely fails to decide the question.

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[1] The green liquids often vomited from the stomach claim, by turns, at least two, rarely a third, of the sources above referred to. Any transparent liquid

Flatulence, as a symptom of gastric disease, occupies a somewhat equivocal position, though one which is every way too important to be overlooked.

I may remind you that, in speaking of the gases found in the stomach and intestines, incidentally to the subject of 'Digestion' in the Physiological Course, I laid before you reasons for coming to the following conclusions respecting their sources.

1. Air is habitually introduced into the alimentary tube from without the body:—by some persons, voluntarily, as an act of deglutition or eructation; by all persons, in the ordinary act of swallowing, probably in a state of mechanical adhesion to the bolus of food, and certainly in a state of minute division in the frothy saliva. The air thus introduced into the stomach undergoes a process of diffusion or interchange with the elastic fluids dissolved in the blood of the gastric capillaries: a diffusion which converts the ordinary gaseous mixture of the atmosphere into one containing less oxygen and more carbonic acid, and in a degree varying according to the duration of its sojourn in the stomach.

2. Gases are developed in the alimentary canal by the decomposition of the food it contains.

There can be little doubt that the gases of the intestines are chiefly due to this origin; and that even the healthy stomach generally contains, mixed with its gaseous contents derived from the source already specified, a portion of aeriform fluid referable to this origin. The metamorphoses which constitute the digestive process are so easily susceptible of being carried a

possessing this colour will generally be found to derive it from bile: darker green hues being quite as frequently ascribable to changes undergone by this secretion subsequently to its reaching the cavity of the duodenum or stomach, as to its original characters while yet contained in the liver and gall-bladder. The dark green *flocculi* of such vomited liquids are also sometimes biliary. But they are, much oftener, altered blood: a change of colour which, in my opinion, generally implies either a moderate quantity, or a long delay, of the hæmorrhagic effusion within the stomach, often both of these conditions simultaneously. I may, however, surmise from one or two necropsies that both the watery and the flocculent green vomit exceptionally derive their colour from a transudation of the colouring matter of the blood through the congested vessels, without any escape of corpuscles. The alimentary admixtures capable of giving rise to a similar colour are both too numerous and too obvious to mention. (Compare the remarks on this subject in the lecture on Gastric Ulcer.)

step further, and thus of giving off more or less gaseous fluid, that, though it is one of the offices of the various digestive juices to repress and prevent all decomposition or putrefaction (in the strict sense of these words), some fraction of the food generally escapes their influence, and distends the stomach with a mixed gas, the large hydrogen ingredient of which is conclusive evidence of its having some other source than any mere deglutition of air. Indeed, under the influence of the heat and moisture of the digestive cavity, it is obvious that nothing short of the completest adjustment—both as to quantity and quality—of the various secretions to the several constituents of the food on which they operate, could be expected to prevent such a process. And, practically, the examination of the healthiest living individuals always shows a sonorous response of the gastric region to percussion; such as, taken in conjunction with the analyses of Magendie and Chevreul, leaves no reasonable doubt, that the term "flatulence" is one of degree, and not of kind; and that the question, whether *flatus* is present in abnormal amount in any given case, depends (like so many other questions involving health and disease, not to say good and evil) on the *poco piu e poco meno*, which govern so many of our decisions in actual life.

3. Another source of flatulence suggests itself in the case of various constitutional diseases: namely, the evolution of gases by the decomposition or putrefaction of fluids derived from the organism itself. That gases may be thus evolved in cavities of the body seems established by various authentic cases of *physometra*; where the elastic fluids which distend the uterus can only be referred to such a source. And, from analogy, it seems probable, that, in numerous disorders known to be attended with a peculiar proneness of the fluids to putrefaction, the spontaneous decomposition of the ingesta contained in the digestive tube, favoured by heat and moisture, and unchecked by the action of healthy digestive juices, is accompanied and furthered by a kindred decomposition of the depraved and altered secretions themselves. Such a variety of flatulence, however, takes but an infrequent (as well as small and contingent) share in the production of this symptom in the proper gastric diseases we are now about to study.

4. Lastly, as regards the evolution of gases in the digestive tube, by a process of secretion from the vessels of its walls, I may remind you that, though various authors regard such a process as taking a large and important share in the production of flatulence, a careful inquiry into the subject has led us altogether to deny its occurrence. To say nothing of the complete inapplicability of the term "secretion" to a process which, if it occurred, would, from all analogy, constitute an act of diffusion of the same kind as that which obtains in the lungs and skin—the facts hitherto ascertained concur to disprove even this qualified evolution of these gases from the blood. The gases found in the intestines are, both in quantity and quality, precisely those which would be evolved by the decomposition of the various substances used as food; and under circumstances of complete and sudden starvation, are often entirely absent from a great part of the alimentary canal. Some of them—such as hydrogen, carburetted hydrogen, and sulphuretted hydrogen—have never been detected in the blood in any such appreciable quantity as would be necessary to explain their evolution from it. Nor can any parallel to such a gaseous excretion be found even in the case of those structures which (like the lungs) are specially organized with reference to the giving out from the blood of certain of its gases, and the taking up of others from the surrounding air. For the gases just alluded to, as absent from the blood, are equally deficient in the air of expiration: nay more, involve, directly or indirectly, a deoxidation of water, such as is not only without parallel in the chemistry of the organism, but is curiously opposed by that oxidation of hydrogen which forms about a pint of water daily in the healthy human subject. While the carbonic acid and nitrogen common to *flatus* and expiratory air are scarcely less distinguished by their quantitative relations,[1] than are these others by their presence and absence respectively.

One argument in favour of the secretion of gases by the digestive tube must, however, be recalled: namely, that deduced from a well-known experiment by Magendie, in which the deligation of empty intestine in a healthy dog is soon followed by its distension with flatus. The inconclusiveness of this argument will sufficiently appear, when I point out that the experiment does *not* exclude all alimentary matter; but that, on the contrary, since one grain of starch or sugar would yield, by decomposition, gases capable of occupying about eight cubic inches of space, no such experiment can exclude the presence of sufficient food to account for the gases of the resulting distension:—can approach, indeed, the trustworthiness of the contrary observation, as to the empty and contracted state which often results from starvation.

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[1] In round numbers we may estimate that intestinal flatus contains ten times the quantity of carbonic acid, and two hundred times the quantity of nitrogen, exhaled from the blood in the air of expiration.

Hence, in respect to flatulence, as a symptom of gastric disease, we may start with these propositions. That the stomach and intestines generally contain a certain quantity of aeriform fluids, derived, in great part, from the decomposition of ingesta. That it is only where they are excessive and troublesome that their presence is strictly abnormal. And that, among the causes of such an abnormal amount of these gases—in one word, of flatulence—the most immediate and obvious are, (1st) a quantity of food which is too large, either absolutely or relatively to the digestive juices of the individual; and (2nd) a quality of food which (either from existing or nascent putrefaction, or from a peculiar proneness to it, or even from a peculiar composition[1]) favours this change.

The pathological import of flatulence is, however, by no means summed up in these general statements. And the diseases of the stomach and bowels offer us certain phenomena, by which we are in some sense bound to test the conclusions we have come to against the alleged secretion of gases from the gastric or intestinal blood. Not only are there various abnormal states of the digestive canal, in which flatulence occurs under circumstances at first sight suggestive of no very close relation to the above laws; but we occasionally meet with cases in which this symptom appears and disappears with such startling rapidity, and seems so completely independent of every other abnormal condition, that it is no wonder if a superficial observer or lax reasoner should think the secretion of gases an absolutely unavoidable explanation. A person, for example,* is prevailed upon to break a fast of many hours with a morsel of some kind of food which he habitually digests with difficulty. He is instantly seized with acute pain in the epigastrium; but, before his alarm and agony can obtain any medical relief, is restored to complete health by a copious eructation of flatus. The advocates of this secretory doctrine would, perhaps, think such a case susceptible of only one explanation: that the empty stomach had been the seat of a sudden and copious evolution of gas, producing distension and pain, which ended by the expulsion of the *flatus*.

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[1] A cause which appears to have some efficacy in the well-known effects (both in Man and animals) of various articles of food (*e.g.*, peas, beans) rich in protein-compounds which contain much sulphur, on the evolution of sulphuretted hydrogen.

But there is no necessity for accepting any such explanation. On the contrary, the physiology of the digestive canal not only justifies, but demands, a very different interpretation of the facts.

To begin at the beginning, let me remind you that the bulk of an aeriform fluid is dictated by the pressure to which it is exposed: so that, for example, to double the pressure of a cubic inch of gas would diminish its bulk to one half; or, conversely, to halve its pressure would convert one cubic inch of bulk into two.

Now within certain limits (dictated chiefly by the general pressure of the atmosphere), the intestinal gases are exposed to the influence of two varieties of pressure; which, while they seem chiefly or exclusively to affect the bulk occupied by a given amount of *flatus*, often probably concur with a slower alteration in the quantity developed in the digestive canal. The pressure of the abdominal muscles on the intestinal canal, and that of the muscular parietes of this tube on its contents, thus to a great extent regulate the bulk of the aeriform fluids by which it is so largely occupied. It is chiefly by diminished pressure that the most characteristic effects are produced. And their pathological significance depends mainly on the mechanism of this diminution; namely, a muscular relaxation in answer to a stimulus. Thus, when we irritate the peritoneal coat of the intestine in a living animal, we excite a local relaxation (a

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[1] For obvious reasons I select a striking illustration, but the above case came under my own observation many years ago.

kind of bulla, or bulging) of the wall of the canal:—a swelling formed by the relaxed coats of the bowel, and occupied by its gaseous contents. In the experiment of Magendie alluded to, it is probable the distension observed was partly due to a more general effect of this kind, brought about by the irritation of the ligature. And it would be easy to adduce a variety of morbid conditions of the alimentary canal to illustrate the propositions:—that relaxation of this kind is connected rather with irritation of the peritoneal, than of the mucous coat; of the trunks, rather than the periphery of nerves: and that (*cæteris paribus*) its degree and extent vary with that of the irritation; so that (as exemplified by severe peritonitis), in its more characteristic forms, it engages, not only the muscular wall of the intestine, but those muscles of the belly which we have seen[1] to be really co-ordinate with it, and thus produces that general gaseous distension of the flaccid (and so to speak, paralysed) abdomen, which constitutes some forms of tympanitis.

It is scarcely necessary to insist upon the essential distinctions between the more characteristic forms of tympanites and flatulence; between a merely increased bulk and a really increased amount of *flatus*; between that which is not, and that which is, expelled by the contractile force of the two muscular tissues—abdominal and intestinal—that enclose the contents of the bowel. But there can be little doubt that, in many cases regarded as mere flatulence, it is impossible to exclude that reflex process of relaxation in answer to stimulus, of which the tympanites of peritoneal inflammation affords the best illustration. Especially is this the case with those temporary and local accumulations of *flatus* of which we are considering an example. Here, for instance, instead of a sudden secretion of gas, causing pain and distension of the stomach, and ending in an expulsive act, I should offer a very different explanation:—that the irritation of the morsel of unwholesome food caused a relaxation of the gastric coats; that the pylorus (generally patulous to the non-alimentary contents of the duodenum,[2] and specially so in virtue of this relaxation) allowed the rarefied gases of the stomach to be increased by an addition from the duodenum; and that the resulting eructation expelled a fraction of the total gaseous mass. That, under these circumstances, expulsive contraction should follow relaxation is not surprising; indeed, you will recollect that, in the normal rhythmic peristalsis of the muscular wall of this canal, these two states have exactly the same sequence, both in time and place.

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[1] "Cyclopædia of Anatomy," art. "Intestine." Supplement, p. 370.

[2] *Op. cit.*, pp. 315, 319.

You will observe, however, that such a view, which rests, not only on its own inherent probability, but also on that disproof of the secretion of gas by the digestive tube which we have established, suggests considerable doubt as to how far the pain is caused by the flatulence. And a careful analysis of the incidents of such a case will quite confirm this doubt. Without going so far as to assert that distension does not often materially add to the pain of a flatulent attack, we are at least fully entitled to conclude, that (far from pain being always preceded and caused by flatulence) in this chain of abnormal phenomena, pain, relaxation, and contraction or expulsion, are generally three successive links; and that not only may the first occur, without always or necessarily calling forth the second and third, but that it is essentially the exponent of an irritation, itself the cause of all three.

LECTURE II.

Circumstances connected with the Examination of the Stomach after Death—Gastritis.

Before entering upon the diseases of the stomach, I purpose to notice some points connected with the examination of this organ after death;—points which we must understand, if we would rightly appreciate its morbid anatomy in the several maladies we have to discuss.

The history of anatomy, as well as of pathology, furnishes numerous examples of the extreme importance of a full and careful study of the processes of death and putrefaction, such as, in the Lectures on Forensic Medicine, I consider it my duty to submit to your notice. The gaseous contents of the arteries after death led the ancients into an error (as to the fluids these tubes transmit during life), which is still recalled by their name. The solid condition assumed by the adipose tissue of many animals after death, has doubtless often suggested strange mistakes as to the phenomena of their muscular (and especially their cardiac) action. The opaque and solid nervous tissues of the dead body have probably undergone a similar (though less marked) change of physical characters before they are seen by us. And the serous effusions found in certain cavities, as well as the fibrinous clots met with in the larger vessels, have notoriously been mistaken for products of disease, instead of the mere incidents of death, which they are now known generally to constitute.

Of all the organs of the body, perhaps there is not one of which the successful examination is opposed by more difficulties than the stomach; not one in which death introduces changes of greater extent, variety, and rapidity, or more closely simulative of the effects of disease. So much is this the case, that, in our knowledge of its healthy anatomy, the stomach has long been (perhaps still is) considerably in arrear of most other organs. The ordinary interval which our reverence for human life very properly interposes between death and dissection—between the parting of soul and body, and the throwing open of the untenanted mansion to our gaze—suffices, in almost all instances, seriously to alter and damage many of the gastric structures. And any circumstances which aid to keep the body at a higher temperature than it would otherwise assume, and thus postpone or protract the process of its cooling after death, further increase this impairment of the tissues of the stomach. So that, for example, the examination of a corpse which has lain from ten to thirty hours in an atmosphere of about 60° Fahr., rarely affords us a stomach sufficiently perfect and entire to exemplify the structure of the healthy organ during life.

It may be premised that the organ itself has some inherent difficulties, such as may be traced in a microscopic examination of the freshest and healthiest specimen. Its cell-growth is so delicate as to be readily damaged and burst by the endosmose of water, or other fluids of low specific gravity. The peculiar mutual attachments of its structures make it very easy to deform and distort them to an unusual degree. For example, the elastic basement membrane of the gastric tubes is inseparably united with the matrix at their mouths, but easily loosened and dislodged at their blind extremities. Hence, in tearing up specimens for observation, the former parts of these tubes are often destroyed altogether; and the latter forced into such a state—so twisted, bulged, or even broken or rolled up—as easily to mislead the observer, when he sees them apparently isolated and uninjured in considerable lengths. It would be invidious to specify the errors (both in pathology and anatomy) to which such simple facts have given rise.

In tracing the dead stomach towards its decomposition, you will do well to bear in mind that the clue to all the changes detailed, lies in a very simple proposition respecting this organ. It has (so to speak) a general and a special set of changes: one which belongs to it as to a soft part, composed of much blood and muscular substance, and arranged so as to afford a large surface of contact with the aeriform fluids it encloses; and another, which is connected with its function, as an organ which secretes, by means of two peculiar cell-growths, a liquid which has the specific property of dissolving protein-compounds. The former gradually permits the organ to undergo the physical and chemical changes which constitute ordinary putrefaction, the latter adds, to this form of dissolution, one effected by the powerful gastric solvent. The former yields as its products the new compounds usually disengaged by putrefaction: especially ammonia, carbonic acid, water, sulphuretted and phosphuretted hydrogen, &c. The latter forms a specific liquid singularly resistant of putrefaction—the peculiar solution of hydrated protein-compounds called peptone. With great differences, as we shall see, in the nature and rapidity of these two factors of gastric dissolution after death, they concur in a variable degree in every stomach we examine by a necropsy.

First after death comes an engorgement, which specially affects those (*quasi* venous) capillaries occupying the free surface of the stomach, and surrounding the mouths of the gastric tubes. (*r*, Fig. 9, Fig. 10, p. 23.) In the stomach, as in the rest of the body, it is probable that the arteries empty themselves into the capillaries by the contraction of their elastic and muscular walls at (or immediately after) the moment of death. The capillaries almost as quickly propel their contents onward into the adjacent veins, which become correspondingly distended through a variable extent of their course. Which of the veins are thus engorged, and to what distance their different degrees of distension extend onwards toward the heart, or backwards towards the capillaries, must depend on a variety of circumstances, in which the mode of death, the state of other organs, &c., are all intimately concerned. As regards the stomach it need only be said, that this engorgement specially affects the vessels already mentioned, and the digesting organ; that it occurs a very few minutes after death; and that, whether from structural and functional peculiarities to which I must not now allude, or from the mere muscularity of this part, it specially avoids the pyloric region or sac.

It is impossible to draw an exact line of demarcation between this speedy congestion (apparently engaging the stomach both as an internal organ and as a secreting structure) and the true *hypostasis* which brings about discolorations like those seen on the exterior of the body ten or twelve hours after death. For not only do they merge into each other by inconceivable gradations of time and amount, but it is the very same blood that occupies the gastric capillaries as an effect of this congestion, which gradually obeys the impulse of gravity, and descends to a greater or less extent towards the lowest or inferior surface of the organ. Such a true hypostasis or gravitation is, however, less complete (and I think somewhat later) in the stomach than in the lungs or the subcutaneous tissues.

Other indications of the dominance of physical laws, heretofore subordinate to the living forces, may be seen in the appearances of infiltration or imbibition the stomach so often exhibits. In this organ, the solution and transfer of the colouring matter of the blood are not easy to verify (though they doubtless occur) in the presence of such marked changes of colour as are brought about by other causes. But the imbibition of biliary colour, as well as of the various hues derivable from food or medicine—such as the red colour of wine or logwood, or the black colour of the metallic sulphurets reduced from the solutions of their salts—are familiar instances of this kind of infiltration, greatly modifying the aspect of the organ.

Here is perhaps the place to allude to some other peculiarities, in great part due to the casual circumstances which precede or immediately follow death. The shape and size of the stomach, and the thickness of its coats, as seen in necropsies, are scarcely less variable than its colour, its consistence, and the degree of its congestion. As regards size and thickness, not only do they vary in different individuals, as do such physical circumstances in all other parts of the body, but it must be especially borne in mind that, in an organ constructed like the stomach, the two appearances are mutually convertible into each other. A dilated stomach, for example, is laid open by an incision, and speedily (with a degree varying according to the amount of contractility left by decomposition) contracts into a state perhaps as simulative of hypertrophy, as its previous condition was of dilatation. Hence the real degree of either of these states must be determined as a balance of the two; must be estimated, for example, by observing the hypertrophy which co-exists with moderate distension, or *vice versâ*. In like manner, unusual length or width of stomach are often illustrations (occasionally permanent, more frequently temporary) of a gain in one dimension being tolerably compensated by a loss in the other, with little real deviation of size. And of course it is not merely the casual condition of the stomach at death, with the circumstances of its *rigor mortis*, which regulate its size or the degree of its contraction, as seen in a necropsy; but also (and in an even greater degree) the state of habitual distension which has preceded death—a distension often essentially independent of the stomach itself. Thus starvation, vomiting, heart-disease, emphysema, and stricture of the œsophagus during life, may all give rise to a small or contracted state of the stomach; the single fact in which they sometimes concur (the absence of the normal distension produced by food) giving rise to this state as its physical result. In all, too, a slow and careful process of artificial dilatation will generally show the true character of the appearance.

What is called hour-glass contraction of the stomach is occasionally a congenital peculiarity in the shape of the organ, as its situation and appearance would suggest. Oftener, however, it also is a mere phenomenon of the final contraction or *rigor mortis* of the stomach. The two are easily distinguished by inflation, no reasonable amount of which suffices to remove the congenital constriction. But I am persuaded, from careful inspection of specimens, and analysis of their histories, that many cases supposed to be congenital contractions of this kind, have been the results of a cicatrized and contracted ulcer.

The state called mammillation of the stomach is another effect of the contraction of the muscular fibres of this organ. I am aware that some good authorities still regard this as a morbid condition, produced by an accumulation of glandular or fluid contents within the gastric tubes. But I have convinced myself that this view of its nature is untenable. For it not only occurs in persons dying suddenly during health, but also in stomachs otherwise perfectly healthy. It is rarely accompanied by any change in the bulk or appearance of the secretory structures. It affects chiefly the more muscular pyloric half or two-thirds of the organ. Its projections often merge, by slight gradations of size, into wrinkles closely akin to those formed in the healthy and contracted organ. It may always be effaced by pressure, generally by prolonged dilatation. And lastly, though, when of limited extent, it is compatible with a relaxation of some other part of the stomach, it is generally associated with local contraction, and is never found both marked and extensive, save in a contracted stomach. As respects the mode in which it is brought about, I believe it to be due, not to a mere contraction of the longitudinal and transverse layers of the general muscular coat—as is evidently the case with the ordinary *rugæ* (see p. 14)—but to a much more irregular and uncertain contraction specially shared in by the various bundles given off from this coat to form the muscular constituent of the matrix (see p. 17), in which the tubes of the mucous membrane are packed vertically side by side. In short, it seems to be somewhat analogous to those curious distortions by which the villi share in the *rigor mortis* of the muscular coats of the intestine. I may add, that the permanence of the phenomenon seems to be sometimes furthered by a subsequent process of transudation, which distends the submucous areolar tissue of the various isolated projections or *mammillæ,* with a serous fluid of the same kind as that often found occupying a larger extent of the cellular or areolar coat. (See p. 76.)

With those re-arrangements of the blood, vital and physical, already mentioned—with this congestion produced by the phenomena of death in the three classes of vessels, and the transudation and gravitation which succeed them—concur two other processes (one of effusion, and one of solution or digestion) a brief mention of which may end these allusions to the ordinary course of dissolution in the stomach. In respect to the date of their occurrence, doubtless both of them commence almost at the moment of death, although there is rarely any marked evidence of their presence until the changes already described have taken place. As to the frequency of their occurrence, I believe that neither of them is ever completely absent, no matter what the causes or circumstances of death. As to their nature, it is doubtful whether the effusion occurs incidentally to the process of transudation already mentioned—and thus consists of a serum which was formerly contained in the vessels, and which, in permeating the gastric cell-growth, carries away certain of its soluble ingredients—or whether it is a true secretion analogous to that poured out during life. It is equally doubtful how far the nature and amount of this effusion regulate or determine the process of solution undergone by the gastric coats. But the following remarks may partially describe what really takes place.

Nothing is more common, in many of the healthiest animals slaughtered as food, than a kind of desquamation or exfoliation of the columnar epithelium of the gastric ridges. In the Pig, for example, the whole stomach may sometimes be seen to have shed this coat as early as two or three hours after death; so that what appears to be (but is not) the true surface of the organ, is covered by a copious dense mucus, the true nature of which is at once revealed by a microscopic inspection.

An occurrence nearly analogous to this often occurs in the human subject. For example, in a young adult dying of some acute disease, even a speedy necropsy, in cold weather, sometimes shows a similar superficial mucous *exuvium* covering the whole organ. And a careful microscopic examination of such a stomach proves that (together with what, for obvious reasons, is generally a far greater amount of congestion than in the stomach of the comparatively exsanguine slaughtered animal) there is much the same destruction of surface, associated with equally little change of the deeper parts of the gastric tubes, and of the pepsinous cell-growth they contain. From this superficial change—in most, if not all cases, a combination of desquamation, effusion, and solution—commences the *post-mortem* digestion of the stomach; which gradually sinks throughout the whole depth of the mucous membrane, and combines, in innumerable gradations of admixture with the vascular phenomena already mentioned, to produce results so complex, and so variable, that we may fairly say no stomach we see is precisely like any other.

As a rule, however, it may be alleged, that the younger and healthier the subject of examination, and the more acute the disease which has caused death, the more rapidly and effectively does this exfoliation and dissolution take place. It often proceeds with so much uniformity and regularity, and the laminæ of mucous membrane it strips off leave so level and so smooth a surface, that its extent would pass quite unsuspected without a careful examination of the organ. In other cases it specially affects the summits of the folds into which the mucous membrane of the stomach is thrown by the contraction of its muscular coat. Great congestion seems to disfavour[1] it; not only by the additional materials it submits to the solvent process, but also (I believe) more specifically. It brings, too, a set of complications all its own. The solution of the blood thus attacked is preceded by changes of its colour and consistence, precisely analogous to those already mentioned (p. 61) in connection with hæmorrhage into the stomach during life. And hence we not only find a variety of shades of brown, black, or slate-colour, diffused more or less widely and regularly over the organ, in patches corresponding with the previous congestion; but gradually, as the superficial parts are dissolved, and the deeper attacked, the larger vessels (and especially the large and numerous veins of the submucous areolar tissue) become affected, forming a kind of cloudy brown network in the parts most engaged by it; namely, the cardiac end, and the posterior surface of the organ.

As already stated, it is difficult to determine how far this process is aided by the effusion into the gastric cavity of a serous fluid, such as we find freely poured out in many parts of the body after death. Analogy, however, somewhat discourages such a view, by suggesting that a mucous membrane, with an elaborate and specific office, is an unlikely seat for any large effusion of this kind. And the mere structure of the mucous membrane of the stomach is such as would probably impede and restrain a serous effusion; both by the close packing of the cell-growths which cover its basement membrane, and the remarkable density and firmness of the adherent matrix beneath it. But, while we sometimes meet with cases (especially in dropsical subjects) in which the mucous membrane has a soft and watery consistence, strongly suggestive of a serous infiltration, there can be no question that the looser sub-mucous areolar tissue is often occupied by a fluid of this kind in considerable quantity, giving it a quaggy and humid texture, or even raising the membrane here and there in patches somewhat elevated above the general surface. And on the further decomposition of the organ, the areolar tissue thus infiltrated becomes the seat of a kind of putrefactive emphysema, being distended with gases which crepitate when the coats of the stomach are pressed between the fingers.

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[1] Compare note to p. 81.

In the subsequent changes of the stomach after death, putrefaction resumes (or rather assumes) its ordinary sway: conducting the organ to its dissolution by processes not requiring any special distinction from those which (at a somewhat later period) complete the destruction of the intestinal coats. Why, after first deviating so widely, the dissolution of the stomach reverts to an ordinary type of decomposition, is sufficiently obvious. In a vast majority of cases, there is nothing which at all suggests any considerable process of secretion by the stomach, either at or after death. And hence, as soon as the pepsine already present in the stomach-substance has dissolved so much of the blood or of the tissues as it is capable of taking up, and the solution of peptone thus formed has itself been distributed by a physical process of imbibition in the neighbouring parts, its solvent action comes to an end; and its antiseptic properties are soon overbalanced by the continually increasing decomposition of the tissues it has penetrated. So that, though the solvent force of the gastric structures usually not only softens, but absolutely dissolves, a varying depth of the stomach from its mucous surface outwards, this action often goes no further than to denude the submucous or muscular coats.

Frequently, however, the stomach exhibits proof of a solvent action far exceeding these its customary bounds;—of a process which not only softens, but absolutely erodes and destroys a variable extent of the entire thickness of the stomach, and even attacks the neighbouring viscera.

As a matter of mere description, little can be added to the records left by John Hunter, who first drew attention to this kind of gastric perforation. The aperture it makes in the stomach is an irregular ragged hole, with gelatinous pulpy margins of great tenuity, which break down under the slightest force—a condition often extending over almost the whole of the organ. Of course there are no evidences of inflammation on the one hand, or of putrefaction on the other; the antiseptic action of the gastric solvent remarkably controlling the latter decomposition in most cases. And as a rule, little congestion is present: the process apparently occurring at a date prior to the ordinary hypostasis that follows death, and preventing this change by a specific effect on the vessels or their contents. Hence the whitish or yellowish-grey appearance of these softened and perforated stomachs is a character which aids to distinguish them from ordinary specimens. But, in many cases, even the perforation of the stomach by no means ends this process of solution; which extends to other structures thus exposed to the action of the gastric contents, and softens and erodes the neighbouring liver, spleen, or diaphragm, or even the lung itself, after the perforation of this septum.

In respect to its circumstances, we may first notice, that the ordinary site of this process of perforation is at the cardiac end of the stomach; but that it sometimes varies from this rule by affecting the neighbouring middle segment of the organ, opposite the œsophagus; either anteriorly, posteriorly, or near the small curvature, and with a diminishing frequency tolerably corresponding with this order of succession. The pylorus is scarcely ever affected alone: at any rate, less frequently than is the cardiac end of the œsophagus.

Its other *juvantia* and *lædentia* are equally interesting. As Hunter observed, it especially occurs where a sudden death has immediately followed a meal: in other words, when the stomach contains a large quantity of newly secreted gastric juice. I believe, too, that it is greatly furthered by a solid consistence, and by a starchy or floury character of the food. It is also furthered by the healthiness, and still more by the youth, of the dead subject: as is shown by contrasting its frequency in accidental death, acute disease, and chronic disease; or in children, adults, and aged persons respectively. Of all maladies, phthisis is that with which it is most frequently found associated; though in noticing this fact, we must remember both the frequency of the disease itself, and the comparatively keen appetite which many of its subjects retain to the last hour of life. But it is occasionally met with in a variety of acute and chronic maladies—as in the specimen before us, from a case of pericarditis. And though we are obliged, on the whole, to subscribe to the opinion of Hunter—that injuries of the head form no frequent or special cause of this perforation—yet an interesting case brought forward by Dr. Budd[1] seems to indicate some such causal connection between them, capable of giving rise to the process in the fasting stomach. Lastly, as regards its occurrence in different species of animals, though there are few Vertebrata in whom it may not be often found, its variations appear to depend, less on the mere energy and rapidity of the digestive process, than on the circumstances of this act already hinted at. And as respects temperature, this important element in gastric digestion has all the influence which might be expected (compare p. 30); so much so, that, in the fish ordinarily supplied to our London market, perforation is extremely common in summer, but almost as rare in winter.

If from these facts we would deduce any explanation or theory of this occurrence, we may first observe, that while (as already stated) we rarely open a stomach in which we cannot trace the specific solvent action of this organ upon itself after death, so this ordinary softening and abrasion merge into complete dissolution and perforation by such innumerable gradations, that it is impossible to draw any distinction between them, widely as their extremes differ from each other. That both have a similar cause is therefore an almost unavoidable conclusion:—a conclusion such as almost restricts our inquiry to the various modifying circumstances, or superadded causes, which can increase or diminish, promote or impede, the resulting solution of the gastric coats.

But this conclusion is better deduced more specifically. And when I assert (as I do after years of research) the invariable acidity of the columnar epithelium (*a* Fig. 7) of the stomach, and the fact that the entire gastric mucous membrane of the human subject contains pepsine enough involved in its structures to dissolve about four ounces of albumen—you will see that I am stating what abundantly accounts for a moderate amount of softening and solution in the .stomach after death; at the same time that it suggests that a mere acidulation of the gastric contents, however effected, might suffice to mediate a limited solution and perforation of the stomach, if not also an erosion of abdominal viscera.

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[1] On "Diseases of the Stomach," p. 17.

In like manner I must so far anticipate some unpublished researches as to point out, that there are various causes which can respectively oppose and further the solvent effect of the gastric structure, by introducing modifications precisely corresponding in their results to those which we may observe in experiments on artificial digestion. Especially would I notice, that the protection of the tissues of the stomach against its own solvent juice seems to be partly effected by the secretions—chiefly salivary and biliary — it receives from the neighbouring mouth, œsophagus, and duodenum; and that it certainly secretes no alkaline mucus (I would almost say no mucus whatever) in a healthy state. Hence among local causes capable of preventing gastric solution, we may notice (1), the presence of alkaline saliva and bile in any quantity; and (2), the presence of animal food, absorbing and engaging (in proportion to its minute division of surface) the powers of the pepsinous fluids furnished by the organ. And conversely, among influences capable of increasing the solution produced by a given amount of healthy gastric substance, we may mention (what has often occurred in the cases recorded) a vegetable or starchy character of food: (1st), as offering little substance on which the stomach can immediately expend itself; and (2ndly), as producing by its decomposition, aided by that of the organ and its secretory contents, an amount of acid such as adds the very ingredient necessary for an energetic gastric solution, to a pepsinous fluid itself remarkably deficient therein.

With all this, however, it must be confessed, that the more energetic and complete cases of self-digestion of the stomach can scarcely be explained, except by supposing a true secretion of gastric juice; by a process which, so far as we know, invariably precedes death, and which is almost always connected with its normal immediate cause; namely the presence of food in the stomach. Whether injuries of the head, or of any parts of the nervous centres, can provoke an increased secretion of gastric juice, or can evoke it independently of the presence of food in the organ—or, if so, how far such a result would be comparable to Bernard's well known experiment, in which an injury of the medulla oblongata excites an excessive formation of grape sugar by the liver—are questions to which our existing information affords no answer.[1]

There are many other points of interest, connected with the examination of the stomach after death, which time forbids our considering here. Amongst these I would briefly allude to the pathological import of ecchymosis, and of ulceration, as found in the gastric mucous membrane after death. There can be little doubt, that the first must be regarded as generally indicating, in the stomach, what it certainly does in the subcutaneous tissues—an extravasation occurring during life. In rare instances, however (and chiefly where death has resulted from injuries of the nervous centre, attended with little[2] hæmorrhage), we see minute spots of extravasation under the gastric ridges in animals so perfectly healthy, that it is difficult to avoid supposing these ecchymoses have taken place, either as contingencies of the agony of death, or as a result of those readjustments of the circulation which immediately succeed it. Hence it is possible that slight and irregular appearances of this kind in the human subject are sometimes devoid of all pathological import; especially when they are accompanied by that general venous congestion which we have seen may be present in almost any moderate degree, independently of gastric disease. And in like manner, though a local breach of surface—a loss of substance of the mucous membrane, bounded by a firm and defined margin of this tissue in anything like its normal thickness—can scarcely be accounted for, save as the result of an ulcerative process during life; still with appearances at all less definite, we must sometimes remain in doubt. For example, I have seen instances in which the absence of all marks of reaction around small erosions of the mucous surface has concurred with a somewhat thinned and softened margin of this supposed ulceration, and with irregular softening and slight abrasion of the organ elsewhere, to raise a strong suspicion that such erosions are sometimes chiefly (if not exclusively) due to the phenomena we have traced in the stomach after death. Recollect then, that in this, as in most other departments of pathological (not to say human) knowledge, appearances are sometimes equivocal:—that they require careful study, rational analysis, and, above all, a comparison with the structures of the healthy body after death, and with the symptoms of the diseased subject during life; without which the dead-house will sometimes mislead us as to mere morbid anatomy, and will never enlighten us on that subject to which it should always refer—the nature and treatment of disease.

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[1] The above *juvantia* and *lædentia* of this process of solution are in close accordance with the results of Dr. Powy's researches elsewhere (p. 38) alluded to (compare p. 76).

[2] As in animals killed by pithing.

GASTRITIS.

In a systematic discussion of the diseases of any organ, the frequency and gravity of inflammation, and the rapidity of its course, generally render it the first to claim our notice. In the stomach, however, this malady has, not so much a subordinate rank, as a different import. Rarely met with as an idiopathic disease, it has yet so direct a bearing on the pathology of the organ, and occurs so frequently as the result of the ingestion of poisons, and as an incident of other maladies, that in both a practical and scientific point of view, gastritis has scarcely less interest than pericarditis or pneumonia.

For example, in conceding that acute gastritis is rarely witnessed, save as a result of the administration of poison, we are not in any way excusing ourselves from the study of its symptoms. On the contrary, this very fact points at peculiar and heavy responsibilities in connexion with the malady; responsibilities which require us to be so thoroughly and minutely acquainted with these symptoms, as that the mere physiognomy of an attack of gastritis may arouse our suspicions respecting their cause. And since not even the greatest numerical disproportion is any substitute for a diagnosis, the rarity of the idiopathic disease by no means obviates the necessity for a contrast between the two forms of gastric inflammation. While it certainly suggests some inquiry as to why the stomach is so remarkably contrasted with many other organs.

The gastritis produced by irritant poisoning is, indeed, a model of the symptomatology of the stomach. Shortly after the poison reaches this organ, a gradually deepening sensation of uneasiness or warmth ushers in a more specific pain, soon of an acute burning character. Closely preceding or following this pain comes nausea, rapidly converted into vomiting, and becoming increasingly frequent as the pain augments in severity. The local appreciation of the pain is heightened as the sensation itself becomes more intense; the epigastrium acquiring an excessive tenderness and soreness to pressure; and the patient's body being bent forward, to relax the muscular walls of this region. Not unfrequently the muscles of the belly are spasmodically contracted, as are also various muscles of the extremities. During the accession of these symptoms there is a marked excitement of the system generally; the pulse and breathing being accelerated, and the skin increased in temperature. But this condition is soon exchanged for a state directly opposite, which rapidly prostrates the patient. The skin becomes clammy and cold; the pulse and breathing increasingly feeble. The latter acquires a catchy, intermittent, or even slow character; the diaphragm evidently acting more incompletely and irregularly as this condition advances; until finally, after a variable period of exhaustion, frequently ushered in and accompanied by hiccough, the sufferer dies, having often retained the full possession of his mental faculties up to the last moment of life.

Of course, in the symptoms, as well as the lesions produced by these agents, there is much that is too characteristic or specific of each poison to be included in any such brief outline. The concentration of the particular agent, its solubility, its affinity for water, its chemical action, its solvent effect on the tissues, its constitutional action after being absorbed into the blood, its attraction or determination to a particular part of the canal—can (as you will recal from the Lectures on Forensic Medicine) greatly modify the symptoms just summed up. Hence, quite apart from the results of an analysis, they generally justify a conjecture as to the poison by which they have been produced. The state of the mouth and the œsophagus, the date of access of the pain, its intensity, the nature of the substances vomited, the amount and character of the purging, may thus assist our diagnosis. It is equally obvious, that even the more general or constitutional symptoms require a careful study; and can only be regarded as typical under certain limitations. Thus, the prostration which ends life is often produced by a concurrence of at least three causes: (1) the constitutional action of the poison; (2) the depth or extent of the lesion it has produced; and (3) the destruction of an organ essential to life—three causes, of which idiopathic gastritis, even if acute, would rarely afford more than the last and most chronic. In like manner, analogy indicates (what indeed experience confirms) that the preservation of the mental faculties is often an illustration of the constituent (if we may so speak) which time forms in the abnormal as well as in the normal processes of life; so that, both in the less acute cases of poisoning, and in the somewhat correlative examples occasionally afforded by structural lesions, a slower death from exhaustion is mostly ushered in by delirium ending in coma. A similar law may perhaps be detected in the intense and rapid peritonitis of perforation, whether from corrosive poisoning or from ulceration (see Lecture III). And an analogous, though different, effect of exhaustion may be traced in the comparatively painless interval which often precedes death by acute poisoning, and by idiopathic lesions of the stomach.

The morbid anatomy of poisoning, and of inflammation, can only be contrasted with similar precautions. The former offers us two conditions:—a destruction of tissue, and a reaction of the neighbouring structures. Of these, the latter alone is analogous to inflammation; the former being either independent of this process, or at any rate so greatly modified by the specific chemical agency of the poison, that it may be preferably regarded as distinct from such a reaction.

Among more remote analogies to acute idiopathic gastritis, it would be easy to adduce the effects produced by the introduction of such irritants as foreign bodies, and hot liquids, into the stomach. But, among the numerous instances of this kind recorded, I can scarcely recall one which affords a good example of diffuse or general inflammation thus produced. In the mechanical irritation produced by solid objects (such as hair, false teeth, pins, nails, knives, forks, &c.) we trace what is occasionally a physiological effect, rather than a physical lesion; oftener a mere wound or other injury, giving rise to local inflammation, ulceration, or abscess. Even where the irritant is a liquid, the resulting inflammation may be scarcely less local. Sometimes, no doubt, the mischief is circumscribed by merely casual circumstances: such as we may trace in its occasional limitation to the cardia and pylorus, in the case both of hot liquids and of corrosive poison—an effect which may plausibly be referred to the absence of diluting mucus, and the occurrence of coaptation and friction, at these valves. Where molten lead has been swallowed, the partial and slight character of the injury which sometimes results is probably due to the hot metal having vaporized part of the fluids of the stomach, and thus prevented or deferred its own contact until it has somewhat cooled. Finally, the violent and diffuse injury producible by scalding water is unlike idiopathic gastritis in one important respect:—namely, that there is good reason to believe the resulting destruction of tissue is chiefly due to the effect of the heat itself, rather than to the inflammation it excites;—a pathological distinction, the accuracy and importance of which is well known to surgeons in the case of external injuries of this kind. The remarkable variety of symptoms witnessed in such cases confirms this distrust of their bearing on gastric inflammation.

Hence we start with a kind of typical gastritis;—a mere group of symptoms, obtained by subtracting, so far as contrast and analogy will allow, the casual and the special features of gastric poisoning from the history of this process. If, from a narrative of this kind, we deduct the accidents of the particular case, and the peculiarities of the special poison, the remainder is *acute gastritis*:[1] in other words, not only the outline of gastric poisoning, but the type of acute inflammation of the stomach, and the clue to the symptoms which testify to even less intense and injurious action of this kind.

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[1] Logically, the *genus* of which the narrative is the *species*, and the peculiarities are the *differentia*.

'But is there any such substantive disease as the acute gastritis described by nosologists, or indicated by the group of symptoms thus dissected out of those of irritant poisoning?' Apparently not. And I can only confirm the negative opinion to this effect recorded by Abercrombie and others, by stating, that in all my experience I cannot recall a single instance of acute general inflammation of the gastric mucous membrane, save as a result of poison;—but will add, that the preparations and records of all the cases of this kind I have been able to discover, indicate something quite distinct from any such malady. For, with the exception of one or two cases justifying a suspicion of poison, most, if not all, of them are instances of an effusion of blood, lymph, or pus into the sub-mucous (rarely the sub-serous) areolar tissue of the organ; its other tissues sharing but partially or indirectly in this change. The mucous membrane, often no way involved, appears in other cases to be affected only in a secondary manner; by virtue of the dependence of its vessels on the arteries and veins of the sub-mucous plexus entangled in the effusion. And hence—without going so far as to say that, since the mucous membrane is κατ' ἐξοχὴν the stomach, whatever malady leaves it untouched can scarcely be regarded as essentially gastric—we may at least assert, that any such inflammation is utterly unlike the typical gastritis described by the earlier writers or conceived of under the above symptoms; which, if it ever occurs, must be exceedingly rare.[1]

Eliminating, then, these rare inflammations of the areolar tissue[2] from the subject of acute gastritis, and similarly deferring all consideration of ulcerative disease—both from its partial or limited extent in the organ, and from the doubt that often hangs over its inflammatory origin—we are left without any actual disease of the stomach corresponding with the symptoms of poisoning, and analogous to the acute inflammations of other organs.

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[1] Especially if we consider the sedulous examination which its intensity and fatality almost imply.

[2] See Lecture V.

But it is quite otherwise with the sub-acute varieties of the inflammatory process, which appear to affect the stomach very frequently. And these we may distinguish into two forms. One, in which the malady is caused by a constitutional state, the effects of which are shown in a variety of other organs, as well as in the stomach. Another, in which it is due to causes connected chiefly or exclusively with this organ; which is submitted to an irritative process, somewhat analogous to that typified by the gastritis of irritant poisoning.

The first is well illustrated by the gastric inflammation which attends many *exanthemata,* and which is especially marked in some cases of scarlet fever.[1] Here careful examination, after death between the third and seventh day of the malady, will often show that the inflammation—the vivid red colour, thickening, and ashy grey exsudation—seen on the tongue and fauces, extends through nearly the whole of the digestive canal; and is especially distinct in the stomach, where the casting off of patches or sheets of false membrane is often accompanied by a scanty hæmorrhage of grumous blood. The exsudation consists of cell-growth; in which the healthy columnar epithelium is more or less replaced by cytoblasts (or cells in their earlier stages of development), and is mixed with variable amounts of amorphous albuminous material.

Now though the symptoms present in the acute stage of this malady and its congeners certainly point to the stomach, yet I should hardly feel justified in asking you to accept the few observations on this head, which I have hitherto been able to make, as conclusive evidence that the anorexia, epigastric pain, tenderness, and vomiting, which form such constant features of these maladies, must always be referred to a subacute gastritis, incidental to the *exanthemata.* But they undoubtedly tend toward some such conclusion. And while, in the case of scarlet fever, they are in exact consonance with all that is known of the pathology of this disease (which you will recollect rarely fails to add, to the external inflammation of the skin, and the internal inflammation of the throat, an equally inflammatory process in the independent[1] mucous tract that lines the minute tubes of the kidney), they indicate two points for our practical consideration. One is, that the symptoms of gastritis are perhaps more distinct than they are sometimes supposed to be; at any rate are distinct enough, where any large extent of mucous surface is involved; varying (as the difference of the tissues concerned would entitle us to expect) much more with the superficies than with the depth of the lesion.[2] The other is, that in this particular stage of such maladies, the patient's aversion for food is a wholesome instinct; a rejection of what not only is superfluous and useless, but may be downright injurious, by embarrassing still further the inflamed organ, and risking an increased intensity of the local inflammation, as well as of the grave constitutional symptoms which it is impossible to avoid tracing in part to this gastric origin. Under such circumstances opium may do good; but it is difficult to conceive that stimulants and food (especially of such food as specially invokes the stomach for its digestion) can do the organ itself aught but harm.

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[1] The silence with which this summary of the state of the stomach in scarlet fever has been passed over by some subsequent writers on the same subject, obliges me to notify that it was not only contained in the above lecture delivered in 1857, and printed in 1858, but had been repeatedly committed to the publicity of a large and changeable audience of students during the preceding seven years. [Note to 2nd Edition.]

The other variety of sub-acute inflammation of the stomach is no doubt far more frequent, as well as much more strictly entitled to the term of chronic inflammation. Perhaps the best instances may be seen in *delirium tremens*. As usually witnessed in hospitals, this malady may be roughly grouped under two heads, according as it is produced by acute or chronic drunkenness: by a single long debauch, or by a protracted habit of tippling, ending in the patient's breaking down under a deficiency of proper food, or habitual stimulus. Now, though these two kinds of delirium tremens merge into each other by infinite gradations, their ordinary distinction is very characteristic and important, both as regards diagnosis and treatment. The first is generally associated with a thickly coated tongue, and with an amount of vomiting, pain, and tenderness in the epigastrium, indicative of gastric inflammation; the latter is often quite devoid of such symptoms. The first, in short, is delirium, associated with (and doubtless aggravated by) gastritis; it requires counter-irritation to the epigastrium; it suggests the guarding of any opium which is administered by ipecacuan or tartar-emetic, and the sedulous use of simple aperients; while it contra-indicates (at least such is my opinion) the use of the habitual stimulants in any but minute and largely diluted doses. The second is exactly opposite to it in all these respects.

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[1] I am, however, doubtful whether much stress can be laid upon this adjective ("independent"). Firstly, the careful microscopic examination of the urine generally shows that the mucous membrane of the whole urinary tract down to, and including, the bladder, shares in the desquamation which so gravely affects the kidney, and often with a fairly proportionate severity. And, secondly, there are good physiological reasons for regarding this community of lesion as depending on a natural community of function; as well as on that continuity of structure between the skin and the urinary membrane which seems to be the proximate cause of their special association in scarlet fever.

[2] Compare foot-note to p. 46.

Now if, in the first of these two forms of delirium tremens, the patient dies, what does the necropsy show us? Sometimes patchy, irregular congestion, of varying intensity: occasionally a more diffuse and less vivid colour; occasionally even a scanty extravasation of blood, dotting the summits of the gastric ridges with spots of variable size. And occasionally these hæmorrhages surround points denuded of all epithelial structures, if not occupied by downright ulceration;—which, indeed, unless you examine the body remarkably soon after death, they easily simulate, owing to their being eroded by the gastric secretion. But none of these phenomena are constant. And in some of the best marked cases of this kind I have ever examined, though the mucous membrane has been thicker, whiter, and more opaque than natural, it has been singularly healthy-looking in every other respect, and has afforded me an artificial gastric juice of remarkable potency.

It is obviously possible that in such cases the exsudation usually evidencing the inflammatory state may have been completely got rid of by a kind of desquamative process during life. It is equally possible that the vascular phenomena are materially influenced by the agony of death, and by those readjustments of the circulating fluids which immediately succeed this event. But, on the other hand, mere desquamation or shedding of epithelium is so common an occurrence (both in the stomach of the human subject, independently of all gastric disease, and in animals killed[1] while in perfect health), and the above peculiar pale opaque condensation of the mucous membrane is so unusual, save in this disorder, that it may be conjectured to have some specific relation to the symptoms referable to this organ during life. And if future researches should corroborate this conjecture, we may perhaps explain so singular a variation from the ordinary phenomena of inflammation as due in great part to the effects (local, constitutional, or both) of the alcoholic poison on the stomach itself;—giving rise to irritation, at the same time that it checks and prevents those processes of growth and waste which, however abnormal in the form of an exsudation, doubtless tend to relieve and cure the inflammation that excites them.

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[1] Compare p. 75.

Even the commoner varieties of chronic inflammation maintain somewhat of that relation to the function of the stomach which we trace in these effects of poisoning. Especially does this seem to be the case with those forms of gastritis which are produced by unwholesome food and drink;—a class of cases which doubtless constitute a vast majority of the less acute inflammatory attacks to which the stomach is liable, and which are probably a very frequent cause of some varieties of dyspepsia.[1]

It is to Dr. Beaumont[2] that we owe our most specific information as to the details of this inflammatory process. But for the unique opportunity he enjoyed of watching the changes that occurred from hour to hour in the stomach of St. Martin, it would have been long before the complex appearances seen in the dead stomach would have entitled us to infer the frequency of chronic inflammation in this organ; far less the nature and rapidity of that process by which its symptoms arise, progress, and terminate in recovery.

The evidence of inflammation he thus observed seems quite complete and incontestable. The pale pink colour natural to the mucous membrane of the healthy stomach was exchanged for a somewhat livid erythematous redness; which was distributed throughout the organ in irregular patches of various sizes, and in its most intense form amounted to a kind of ecchymosis or extravasation of grumous blood. Secretion, too, was arrested; as was shown by the want of all flow of gastric juice on the introduction of food, as well as by the complete absence of mucus in the fasting state of the organ. Lastly, he noticed what, in inflammations of mucous membrane, is tantamount to exsudation: namely, an overgrowth of epithelium, forming patches of false membrane, which, often very well marked at the edges of the erythema or extravasation, were elsewhere visibly distended (or separated from their basement membrane) by a puriform fluid, giving them the appearance of sharp-pointed, white pustules. The subsequent abrasion of these patches or pustules of course set free their muco-purulent contents in the cavity of the stomach, as well as the rolled-up shreds that had previously enclosed them. These local changes in the stomach were accompanied, in their worst degrees and earlier stages, by pain at the epigastrium and sternum; nausea; a furred tongue; headache; vertigo; loss of appetite; constipation; and soreness or tenderness at the pit of the stomach: the latter symptom often lasting longer than any of the others. But in slighter degree, they were almost unattended by any symptoms whatever. And even when tolerably well marked and persistent, they were not incompatible with a tolerable (or even craving) appetite.

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[1] See Lecture VI.

[2] *Op. cit.*

Such characteristic evidences of inflammation, taken in connexion with the above symptoms—and especially with the fact that the process may and does occur without symptoms at all—naturally suggest that much of the dyspepsia met with in practice is to be referred to what is thus, in every sense, chronic gastritis. More than this, however, we are scarcely entitled to assert on the slender (though valuable) materials now at our disposal. That it is not merely in alcoholic poisoning, or in the dyspepsia of drunkards, such a process takes place, is sufficiently shown by Dr. Beaumont's multiplied observations; where it is recorded as having occurred, on different occasions, with scarcely any variations but those of intensity, from various causes:—excess in respect of alcohol; of food; of exercise after meals; of condimentary substances (mustard); and lastly, of mechanical irritation, incidental to his experiments. As some of these are precisely the causes to which we shall hereafter trace ordinary dyspepsia, they strengthen our suspicions that this malady is often due to a similar process. But at present we are scarcely entitled to any definite and general conclusions of this kind; the less so, that we shall find some varieties of the malady appear to claim a different origin.

Indeed, we must be careful not to strain such analogies too far; or rather, not to regard one class of them too partially or exclusively. Even in this very alliance of gastritis and dyspepsia, which is established by Dr. Beaumont's observations, and is confirmed by experiments on animals,[1] we may trace something more than an analogy to the effects of the less powerful irritant poisons;—from which, indeed, however we classify them, we should find it difficult to exclude such substances as raw alcohol, or even mustard, pepper, and other condiments. In short, we come to something like a general conclusion with respect to inflammatory affections of the gastric mucous membrane; namely, that whatever may be those modifications of the process of inflammation in this tissue, which our means of investigation do not allow us to appreciate, distinct and satisfactory evidence of its occurrence is almost limited to cases in which a direct irritation of the organ has been either the remote or the exciting cause. In other words, all the more obvious phenomena of gastric inflammation seem strictly incidental to the function of the organ;—a statement for which we might perhaps find a parallel in true hepatitis and nephritis; and for which we have but a doubtful contrast even in the case of such comparatively lowly organized and inactive structures as serous membranes and areolar tissue.

Hence, then, it is only in a very restricted sense that we can speak of the stomach as enjoying almost an immunity with respect to acute inflammation, while extremely liable to the sub-acute or chronic variety of the process. While there may be varieties of both acute and chronic gastritis, the rarity and obscurity of which have allowed them hitherto to elude our research, the two forms with which we are acquainted appear to merge into each other (both in respect of their pathology and symptoms) by infinite gradations. And although it is impossible to deny the great distinction between an excessive meal and a dose of corrosive sublimate, or to doubt that, by defining what is relatively excessive, the state of the constitution is really a cause of the resulting chronic inflammation; still the circumstances of digestion—the mechanical and chemical changes the food should undergo, and the spontaneous decomposition which mere deficiency of secretions can permit —go far to explain how the constitutional or remote cause may bring about a local or immediate one, in the shape of a gastric irritation.

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[1] As in Dogs with gastric *fistulæ*.

Any more detailed explanation of the scanty share taken by idiopathic inflammation in the maladies of the stomach, would perhaps be out of place here, as it certainly would be premature in the present state of our knowledge. But we have already alluded to the rapidity with which all evidence of the process is removed, owing to the effusion of its products on a free mucous surface. And the contrast afforded by inflammations of some other organs indicates how this favourable position of the gastric membrane not only wards off a variety of perils, but absolutely tends to check or prevent a continuance of the inflammation. Just as, in bronchitis, a patient runs an imminent risk of suffocation from the mucus poured into his respiratory organ; or, in nephritis, is in danger of uræmia from the analogous obstruction caused by an inflammatory effusion into the minute tubes of his kidneys;—just as in hæmoptysis and renal hæmaturia, the mere effusion of blood sometimes provokes, or at least augments, inflammation—so, conversely, we may conjecture that, in the digestive canal, the speedy removal of all effusion often limits the inflammatory process itself.

Whether any part of this immunity is specifically gastric, and is connected with the solvent action of the stomach on the protein-compounds, I hardly dare to inquire. But we may bear in mind the density of the matrix in which the cylindrical stomach tubes are packed side by side; and the repletion of these tubes with a twofold cell-growth, such as allows no exsudation from the surrounding capillaries to reach their minute calibre, save through a thick mass of pepsinous cells, and a thinner but more solid layer of smaller cells. And while, on such a structure I would found a plausible conjecture (remember, a conjecture only) why inflammatory exsudation rarely involves the whole mucous membrane, I cannot forbear adding, that careful researches entitle me to conclude that, in all the more sub-acute varieties of gastritis it is the free mucous surface—the inter-tubular ridges, the stratum of columnar epithelium belonging to them and to the open mouths of the tubes, and the subjacent vessels—which the inflammatory process chiefly, if not exclusively, affects.

Whatever the cause of this immunity, there can be no doubt as to its effect; or as to the extreme importance of an organ like the stomach being little liable to any profound or general disturbances, such as an acute gastritis would involve. Dangerous and barren as are all speculations on "final causes," when we find a complex (and at least dimorphous) glandular structure (the destruction of which would bring with it the loss of a function essential to life, and incapable of replacement by any other organ of the body) protected against a process which in some other organs (as, for example, the skin) is neither rare nor dangerous, it is surely legitimate to comment upon the value of the arrangements (whatever they may be) by which this immunity is secured. The more so that (as these imperfect allusions have informed you) in those milder and commoner varieties of inflammation to which the stomach really is amenable we trace a kind of offshoot of its healthy office. That derangement of the stomach which we stigmatise as chronic or sub-acute gastritis, and which really is a wide departure from health, may be viewed from another point of view as a minister of good—as a regulator of gastric digestion; an instrument which, by diminishing or suspending over-taxed or excessive function, and removing or exhausting the results of local injury, affords an important protection, both to the organ and to the organism at large. And we have seen that its details seem to be in exact accordance with such a duty—that it is not only easily provoked, but also that it extends superficially, without increasing in depth; avoiding, as it were, the deeper gastric structures, and thus at once pouring out its products into a cavity, which permits them a speedy dismissal from the body.

The *treatment* of gastritis corresponds so specifically to the nature of the particular case, that a cursory view of it may suffice. Of course the removal of the cause is its first indication: to be attempted, in the acute gastritis of poisons, either by their removal from the organ or by their neutralization there; in chronic gastritis, by the remedies and the regimen which we shall hereafter discuss more fully in speaking of dyspepsia—a disease from which such inflammation is often indistinguishable. The further object of our efforts—namely, that of moderating and limiting the existing mischief—is chiefly to be accomplished by attempting the removal of the more prominent symptoms present. Among the exceptions to such a rule we may notice the vomiting of acute gastritis, which often demands encouragement rather than suppression, owing to its beneficial effect of expelling the poison contained in the stomach. A similar exception may be found in the vomiting which sometimes attends the slighter inflammatory phenomena of a casual surfeit or indigestion.

The remedies most useful in this respect are precisely the most valuable in abdominal inflammation generally. Depletion is only required in cases of great severity; and even here, is best limited to the application of leeches to the epigastrium. Counter-irritation is much more important, being admissible in cases where the contingent general action of a poison, or the exhaustion of the patient, would forbid the abstraction of blood. Here turpentine stupes, mustard poultices, or flying blisters, may sometimes replace ordinary blisters with advantage. Akin to the effect of these is the extreme benefit derivable from continuous fomentations; which, to afford much relief, often require to be quite as hot as the patient can bear them. In the converse application (or introduction) of cold substances to the inflamed stomach, we can scarcely have a better guide for our decision than the feelings of the sufferer as to the relief they afford. Iced water, or small lumps or raspings of ice, seem to allay thirst, and appease pain, even in the severest cases.

As to sedatives, of course the gastritis of acute poisoning must be treated with reference to the particular agent; the depressing or narcotic properties of which often forbid the prescription of remedies whose effects might stimulate and obscure the existing symptoms. In other cases, they are indicated chiefly by pain; and opium (preferably in a watery solution of the extract) maintains its usual supremacy.

In slight or chronic inflammation, the remarks hereafter made in reference to the use of sedatives in dyspepsia may suffice.

The use of aperients in gastritis demands great caution. For the constipation which obtains in acute cases is often almost a provision of nature against a hurtful action. While, on the other hand, all experience indicates that the removal of the secretions and other contents accumulated in the bowels greatly relieves an irritated or inflamed stomach. I believe the best rule is one which steers clear of either extreme, as well as of a mere routine exhibition of agents so powerful for good or evil. When there is reason to believe inflammation has extended to the peritoneal coat, the use of aperients is to be avoided. In other cases, find out (by verbal inquiries, and by a physical examination of the belly) what degree of accumulation is present, and let this guide your conduct as to removing it. If you venture to give the aperient by the mouth, you will of course avoid whatever would irritate the stomach; and are therefore, practically, almost limited to castor-oil. In many cases it is preferable to give it as an enema. And you may secure almost any reasonable degree or extent of action on the bowels by combining, with a full dose of castor-oil, a little turpentine, or even a drop of croton-oil;—a mode of administering the latter energetic purgative, which I have long adopted to the exclusion of every other, because it seems to secure all its advantages, and avoid all its inconveniences and risks.

You will observe that this meagre outline avoids alike the details of treatment suitable for acute poisoning on the one hand, and for inflammatory dyspepsia on the other: forms, in short, an unsatisfactory diagonal between these two converging sides of gastritis. But this mode of considering it (which is all that is either requisite or possible here) is yet strictly clinical. For we do sometimes meet with cases in which the diagnosis and treatment require us to be equally acquainted with either: and ultimately bring us to something equidistant from both. A few months ago, I was consulted by a young man for symptoms of sub-acute gastritis, which had commenced about four days before with catarrh and sore-throat. The epigastrium was exceedingly tender: and was occupied by pain of such severity, that it "drew him double," and kept him in this bent posture, unable either to lie or sit. It was accompanied by frequent vomiting, and by obstinate constipation. A careful inquiry (especially directed towards poison) at last elicited the nature of his case. He was the victim of domestic medicine. A well-meaning friend had prescribed for his cold a teaspoonful of "nitre," to be taken occasionally; and instead of the Spt. Ætheris Nitrici, which formed the traditional prescription, he had taken the powdered nitrate of potash, in doses which a careful comparison showed to be about fifty grains. The administration of castor-oil and opium, followed by warm fomentations, speedily got rid of all his symptoms.

To the foregoing remarks I append a brief notice of three other affections of the stomach; which, in respect of their nature, symptoms, and appearances after death, must be regarded rather as varieties of sub-acute gastritis, than as specific or independent diseases.

Thus *catarrh* of the stomach, often announced during life by little more than indigestion, and indicated after death by evidence somewhat equivocal, offers us, in well-marked cases, phenomena closely analogous to those of chronic or sub-acute inflammation in other mucous membranes. Among its more prominent symptoms are—a feeling of weight or heat in the epigastrium, sometimes amounting to pain, but even then rarely associated with great tenderness to pressure; anorexia and nausea, the latter sometimes developed into vomiting, but oftener stopping short of this act, as a regurgitation of a more or less glairy alkaline fluid; and, with these local symptoms, variable degrees of flatulence and constipation (rarely diarrhœa); together with the ordinary signs of constitutional reaction—among which a furred tongue takes the precedence[1] characteristic to this symptom in gastric maladies generally.

The moderate gastric derangement indicated by such symptoms is rarely or never dangerous to life. And hence it is only the casual death of the patient, from some intercurrent malady, that affords us any opportunity of investigating those changes in the gastric tissues by which these symptoms are provoked.

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[1] A precedence, of course, belonging to it, not so much as a constitutional symptom, as a condition by which the tongue sympathises (often shares) in the derangements of a mucous membrane absolutely continuous with itself.

Necropsies of this kind often show the mucous membrane of the stomach covered, to a variable extent and depth, with glairy mucus closely resembling that regurgitated or vomited during life. And beneath this mucus (which, by the way, varies greatly in transparency as well as viscosity) we find a somewhat thickened and spongy mucous membrane; the increased bulk of which is due, sometimes to a mere serous infiltration (as shown by its pale whitish or greyish colour), but oftener to marked vascular congestion (as shown by its deep red, slaty, or even blackish hue). In other respcets, however, it is quite healthy.

But to each of these three statements I must add one or two details, from my own researches. The glairy alkaline mucus is, in great part, salivary. And though it often affords, to the microscope, a certain quantity of cell-growth, evidently consisting of the columnar epithelium of the stomach, aborted at various stages of its development, as well as (less considerably and frequently) of an admixture of bile; yet I confess myself quite unable to estimate the significance of either of these two admixtures. Recalling to you what has already been said of the shedding of this epithelium after death (p. 75), and reminding you of the natural presence of saliva and bile in the stomach, I may own that the detection of even a larger quantity of such mucus would fail to convince me, that we have here a morbid process of secretion, specific to this disease, and comparable to bronchial or nasal catarrh.

The colour and consistence of the subjacent mucous membrane itself are so various in different cases, that it is scarcely possible to sum them up in one common description. Not only do these stomachs show us every shade of congestion, from the slightest increase of natural colour to a very decided purple or blackish lividity, but the width and depth to which this congestion extends, as well as the abruptness of its demarcation from neighbouring parts, are subject to infinite variety. Sometimes it claims a large proportion of the organ, sometimes is very limited; sometimes it is chiefly pyloric, sometimes affects the more suspicious (compare pp. 71, 76) regions of the cardia and the posterior surface; sometimes it involves all the vessels down to the sub-mucous plexus (p. 21), oftener (as it seems to me) is limited chiefly to the superficial capillaries (*r*, Fig. 9) and their immediate inosculations; sometimes it shades off into a comparatively uncongested (if not rather exsanguine) part, oftener is contrasted by a tolerably marked outline with a generally red surface. Extravasation is rare, even in the most deeply coloured parts: from which, in many cases, moderate pressure partially or completely removes the colour, by propelling the blood into neighbouring vessels. The moist spongy thickening above ascribed to serous infiltration is both less marked, and less constant, than vascular congestion; but, if present, is, on the whole, more regularly diffused throughout the organ.

The otherwise healthy condition of the mucous membrane is, I am aware, a fact which stands in contradiction to the statements of some able observers. But with proper precautions, stomachs which offer well-marked appearances of the kind just described, will often show a tubular structure perfectly normal. And it is my impression that the degeneration and disappearance of the gastric tubes, and the alteration or loss of their cell-growth, attributed to these cases, are essentially the results of circumstances, which we have already alluded to as liable to occur in the examination of the healthy organ, and which are necessarily furthered by the conditions present in these cases:—a statement which may be extended, I think, to some instances of supposed multiplication of the lenticular glands (p. 17).

A careful review of the details just summed up perhaps justifies us in suspending our judgment as to the nature (or even the substantive existence) of gastric catarrh, until larger and more exact information is before us. At any rate, such a study of the malady throws doubts upon its apparent frequency, as seen in the dead body.

Thus, the symptoms during life are common to many gastric derangements, to most forms of sub-acute inflammation, perhaps even to dyspepsias not essentially gastric. And in cases presenting these symptoms we can very rarely confirm our diagnosis by necropsy.

Turning to the appearances after death, we find, that in some of the cases in which they are best marked, there have been few or no symptoms of gastric derangement during life. Against this, however, we must adduce the facts:—(1) that the character of these symptoms would often allow them to be completely outweighed by those of the graver disease of which the patient was dying; and (2) that Dr. Beaumont's observations conclusively show how conditions closely allied to this malady (and even exceeding it in severity) may occur with scarcely any symptoms.

Appearances not preceded by symptoms are deservedly suspected by the pathologist, and certainly have no direct claim on the attention of the practical physician. And when we find some of these appearances doubtfully gastric, while others are at least partially imitated by the processes of death and decomposition, it really becomes our duty carefully to avoid reducing the information they afford us, to a systematic description of a disease. Hence, though, in so far as these appearances transcend those produced by the circumstances which attend and follow death, they must obviously be regarded as morbid, while the mere obscurity or absence of symptoms is at least partially explained as above; yet I would ask you to pause before admitting gastric catarrh to a nosological rank like coryza, or even leucorrhœa. Careful histories of the symptoms of cases, checked by a speedy and sedulous examination after their accidental death—these are the facts which we want, and which perhaps some of you may hereafter have the opportunity of supplying. Waiting the collection of such facts, the subject of catarrh of the stomach is, I think, still *sub judice*.

Hæmorrhagic erosion, a more severe and dangerous affection than the preceding, affords, to a similar inquiry, analogous doubts.

In well-marked cases its symptoms are sufficiently distinct. There is great pain and tenderness in the epigastric region; often accompanied by pain in the back (p. 48), and increased by the ingestion of food; but scarcely exhibiting any absolute intermissions in the intervals of meals. Vomiting is usually both frequent and severe; and often occurs with an empty stomach, or on rising from sleep, as well as after a meal. At variable (in many cases frequent) intervals, this vomiting brings up blood; usually in but moderate quantity; and neither clotted, on the one hand, nor blackened on the other; but copious streaks and patches of blood, mingled with mucus and bile. The appetite is greatly affected; in the exacerbations of the malady, almost suppressed. Lastly, the constitutional symptoms are in most cases proportionately severe; though the constipation, the furred or occasionally aphthous tongue, and the violent febrile reaction of a severe remission, sometimes merge, on its subsidence, into degrees scarcely exceeding those seen in ordinary dyspepsia, and are accompanied by the milder symptoms of headache and flatulence. The malady may last for months, or even years: a duration which (to judge from my own clinical observations) may often be ascribed to the persistence of the habit to which it appears not infrequently due, namely, dram-drinking. It is rarely fatal, except by either inaugurating other lesions in the stomach, or by concurring with some more general malady.

I shall not dwell on the details of the above symptoms, which are subject to many exceptions, as well as to unnumbered gradations. Their significance will better appear by contrasting them with the various corresponding symptoms of gastric ulcer, fully described in my next lecture.

The appearances found after death in characteristic examples like those described above, quite justify the term "hæmorrhagic erosion." On laying open a stomach thus affected, we find it occupied by a variable quantity of blackened or grumous blood, mixed with what is generally a larger quantity of mucus, and (less frequently) with a little bile. Removing these fluids by a gentle stream of water, brings into view a number of minute shallow excavations on the surface of the mucous membrane. These excavations (which exactly resemble ulcers in the fact that they evidently correspond to a true loss of substance of the tissue) have an irregular, but sharply defined margin; rarely elevated above the neighbouring healthy membrane. Their shape approaches a circle or oval. Their ordinary size varies from that of a mustard-seed to that of a pea. Both their base and margin generally have adhering to them a variable quantity of more or less clotted and blackened blood. Traced out under the microscope, they afford indications of being, in most cases, the result of a process, which begins at the inter-tubular ridges (*a*, Figs. 6, 7, pp. 14, 16) of the mucous membrane, and the successive stages of which—congestion, ecchymosis or extravasation, abrasion, and ulceration—may often be seen in different parts of the same stomach. The occurrence of ulceration, or what seems to be such, is apparently followed by its spreading both horizontally and vertically. But in the majority of cases, the superficial extension so far preponderates, that the breach of substance is of much greater width than depth. Still, in many of the best marked specimens, the gastric tubes are destroyed throughout great part of their length; and sometimes appear here and there completely removed. The vessels of the margin of these excavations or erosions are almost always gorged with blood. And this apparently intense congestion, in concurrence with the mechanical results of the lesion in its earlier stages, seems to afford a partial explanation of a peculiar appearance (compare p. 60) which is occasionally seen in such cases; and in which the blind ends of the partially destroyed tubes are found to be filled with blood, suggesting at first sight a specific hæmorrhage from the capillaries surrounding these extremities.

Combining these appearances with the preceding symptoms, it is impossible to doubt that they depict a grave malady; which is often distinguishable in the living subject; and which, in respect to its nature, must be regarded as an ulcerative gastritis. And comparing it with gastric ulcer, its history and symptoms represent it as a variety of ulceration more generally inflammatory, perhaps even more strictly constitutional, than the circumscribed or aggregate form of ulcerative lesion; to which (if I may venture on such an imperfect comparison) it has somewhat the analogy of a severe skin-disease to a cutaneous ulcer.

But while the very same pathological considerations which thus define hæmorrhagic erosion as an important variety of gastric disease, also explain how, in actual practice, both its diagnosis and treatment continually merge into those of inflammation and ulcer respectively, they suggest some doubts as to the nature of many cases which can scarcely be distinguished from it.

We have already noticed (p. 71, *et seq.*) that a considerable degree of congestion is often observed in the healthy stomach after death; and that now and then this congestion—which is often associated with a shedding or abrasion of the columnar epithelium (p. 75) covering the vessels chiefly concerned—amounts to what is really an extravasation (p. 94). Dr. Beaumont's observations have also (p. 91) been referred to, as showing that an amount of hæmorrhage and abrasion very similar to this occurs during life, as a casualty in the history of the stomach; that it is producible by gastric irritation, is evinced by symptoms of dyspepsia, and that (in accordance with the rule—*amotâ causâ, tollitur effectus*) reasonable prudence would often suffice to ensure its complete and rapid disappearance. Similar (but more marked) appearances are occasionally found in the stomach after death from a variety of diseases;[1] the history of the last few days of life also revealing severe gastric symptoms (including vomiting of blood), such as conclusively show the lesions to have preceded death. And though, in most of these cases, the site of the hæmorrhage and abrasion affords, to the minutest inspection, no trace of that loss of substance mentioned as specific to hæmorrhagic erosion; still it must be owned that we do sometimes detect appearances of this kind, and that, even more frequently, the most painstaking observer might hesitate to pronounce a decided opinion for or against the presence of ulceration.

From such analogies, we may pass on to consider the facts on which we have mainly to decide the nature of a very frequent class of appearances in the stomach. In the necropsy of subjects dying from a great variety of diseases, we sometimes meet with appearances suggestive of "hæmorrhagic erosion." A careful inspection shows that many of these cases are disentitled to this term: inasmuch as the mucous membrane is not eroded, but abraded; while both as regards its extent, and the tissues it involves, the lesion is quite different from that which this term would connote. And even among the remaining cases, in which the hæmorrhage seems to be accompanied by a true erosion—by a loss of substance, not merely involving the delicate epithelium, but excavating the basement membrane and its subjacent tissues—there is a large proportion in which the evidence of disease is but equivocal. For the appearance is suspiciously frequent; and is often unattended by any gastric symptoms during life. On the other hand, the presence of a mortal disease, in an advanced stage, may well mask the symptoms of all slighter mischief. And the absence of the characteristic vomiting of blood is often explicable by the considerations (p. 58) which apply to the failure of such evidence of hæmorrhage in other maladies. But, on the whole, the circumstances of gastric decomposition suggest great doubt as to the truly ulcerative origin of these appearances. How far the solvent power of the gastric tissues or secretions may assist to convert abrasion into erosion during life, is a question which is still quite in abeyance. But that some such change is occasionally effected after death seems (to say the least) very probable; though the acceptance of this opinion still leaves us in doubt whether the congestion, hæmorrhage, and abrasion, are not themselves evidence of a condition in the living tissues, differing from true hæmorrhagic erosion in degree rather than in kind.

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[1] Chiefly inflammatory diseases of neighbouring abdominal viscera.

Follicular ulceration, as another variety of the ulcerative process, requires notice chiefly in a pathological point of view. For as a malady of the stomach, discoverable by symptoms, and unconnected with other and more constitutional diseases, it is even less frequent than the foregoing: the appearances which reveal it in necropsies being almost confined to subjects dead of maladies in which the process is certainly but a rare and an exceptional incident. Of all such maladies, phthisis is that in which it is most frequent; and next to this disease come the exanthemata and the acute parenchymal inflammations.

The symptoms of its rare idiopathic cases are sufficiently alluded to by the statement that, comparing it with hæmorrhagic erosion, hæmorrhage is (as we should expect) much less frequent and marked; and that the other indications of gastric mischief (both local and constitutional) are, though irregular, on the whole, less prominent. The principles of its treatment are exactly those hereafter laid down for gastric ulcer.

On examining a good specimen of the lesion, we see a number of minute ulcers, which are scattered over the stomach with variable degrees of closeness; and, as a rule, are perhaps more numerous at the lesser curvature than elsewhere. Their width is usually smaller, and their depth much greater, than the corresponding dimensions of the hæmorrhagic erosions. From this circumstance, their edges are more sharply cut, and vertical; occasionally somewhat prominent;—an appearance which, however, is rarely or never due to any solid deposit. Occasionally, their base and margin have clinging to them some blackened blood and mucus. But generally, the quantity of such mixed liquids is so small, that these stomachs easily clean into the elegant preparations of this kind contained in some of our London museums of pathology: the gastric surface between the ulcers, and the other coats of the organ, being often particularly free from alteration.

Even in respect to these appearances, we may question whether the process of ulceration during life is not in some degree aided by one of gastric solution after death. But it is chiefly in regard to the term "follicular" that pathological anatomy throws a doubt on the accuracy of our existing views. It is possible that (as the nature of some of the associated diseases would suggest) ulceration of this kind is at times dependent on a process akin to that which affects the solitary and agminate follicles of the typhoid or tuberculous intestine:—a process involving the lenticular glands which are the gastric analogues of these follicles. But there are good reasons for supposing that this is rarely the case. Indeed in many instances, the co-existence of healthy lenticular glands, and the shape, size, and depth of the ulcerations themselves, together afford a conclusive negative to such a view. And similar anatomical grounds may be adduced for denying to the ulcerations a "follicular" origin, in the sense of their being specially concerned with the gastric tubes.[1] Observations of this kind have long led me to the opinion, that, even in this form of ulceration, it is on the free surface of the stomach (or the inter-tubular ridges) that the process commences; and that, as regards the majority of cases, we should probably not only gain in accuracy, but avoid a downright error, by adopting an unassuming botanical adjective, and naming them, not "follicular," but "punctate" ulcerations.

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[1] In the anatomical description of both the stomach and intestine, it would be a great advantage if the terms "tube" and "follicle" were attached to distinct

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structures, the latter (as its etymology suggests) being restricted to the closed sac formed by the lenticular or solitary gland. (Compare *Cycl. Anat., Suppl.,* p. 356.)

LECTURE III.

Ulcer of the Stomach.

The disease known under this name may be said to claim precedence over all the other affections of the stomach; uniting, as it does, various features of scientific and practical interest nowhere else found associated. For we can say of it—what we certainly cannot of any other gastric malady—that it occurs very frequently; that it may generally be diagnosed in the living subject; that it runs a protracted course, at any stage of which it may be suddenly fatal; that it is usually curable; and finally, that it is the result of a specific structural lesion such as can be at once detected by an examination of the dead body. Furthermore, it will often be found in practice that the diagnosis of cancer, dyspepsia, or chronic inflammation, requires a process of induction which generally amounts to (and sometimes specifically includes) a reviewal of the phenomena of ulcer of the stomach, before rejecting it as the explanation of the symptoms present.

What I have to say about the *symptoms* of gastric ulcer is derived almost exclusively from two sources:—the records[1] of about 1200 cases, often affording a mere outline of the chief symptoms, but always verified by careful necropsy; and the personal study of more than 200 cases, affording minute details respecting symptoms, but only verified by necropsy in a small proportion of that number. The information derived from these two sources has shown me none but negative discrepancies; in other words, none but differences chiefly explicable by the unavoidable omissions of such brief records.

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[1] In addition to the many hundreds of such records published as scattered cases by scarcely less numerous observers, I have to acknowledge larger contributions of materials from the various metropolitan Hospitals and Museums; and from authors (Cruveilhier, Jaksch, Dietrich, Rokitansky, Dahlerup, Sangalli, W. T. Gairdner, T. K. Chambers, Handfield Jones, Habershon, and others), to whom I have more specifically referred in the monograph, ("On Ulcer of the Stomach," Churchill, London, 1857), from which much of this Lecture is derived.

In what we may regard as typical cases, the history of ulcer of the stomach is made up of the following succession of symptoms.

The malady is announced by disturbances of gastric digestion:—at first, mere uneasiness and pain in the epigastrium; then nausea and vomiting, or regurgitation, expelling the food previously taken, or a tasteless or acid watery fluid. At this stage of the disease, it is sometimes cut short by the occurrence of perforation, ending in fatal peritonitis. Failing such an accident, these dyspeptic symptoms are next complicated by hæmorrhage from the stomach; sometimes a sudden and dangerous gush, oftener a slow and intermittent drain of blood. The anæmia produced by this hæmorrhage is generally associated with cachexia; which seems to be essentially independent of it, being chiefly the result of the inanition necessarily implied by frequent vomiting of the food, or by large destruction of the gastric mucous membrane, and consequent impairment of its function. In young females, another symptom is often present, in the form of more or less complete amenorrhœa, which may be associated with either of these two states of anæmia or cachexia; in other words, may be referred to ulceration, to hæmorrhage, or to both.

The gradual acquisition of all these symptoms conducts the disease, in a variable period of time, to a climax, from whence we may next briefly trace it towards its termination. Retaining the above liabilities to death by perforation, by hæmorrhage, by vomiting, and by exhaustion, the disease may at any time end by one of these modes of dying, or by two or more of them in combination. In other cases, a spontaneous subsidence of the foregoing symptoms, in something like the inverse order of their occurrence, announces a recovery; or a similar amendment is only effected by careful medical treatment, such as quite entitles us to dignify it by the name of a cure. In less numerous instances, these symptoms continue with what is (for obvious reasons) rarely more than a moderate intensity, during a variable period of life; in the course of which their uniformity is from time to time varied by considerable fluctuations of severity. The remissions which form one extreme of such fluctuations sometimes merge into intermissions so complete, that we are left in doubt whether the process of ulceration has merely stood still, or has broken out afresh after the cicatrization of the lesion. In any case, the protraction of these symptoms during many years of life gradually complicates the impairment of nutrition they produce, with that naturally resulting from the approach of old age; mingled with which they then constitute an indirect or conditionating cause of death, the influence of which can scarcely be estimated with any exactness.

But the symptoms alluded to vary so remarkably in different cases, that each of them demands a separate study.

Pain, which is usually the first in the order of occurrence, is also the most frequent and characteristic of them all. Indeed, we may doubt whether it is ever absent from the whole progress of any case. For though there seem to have been instances of gastric ulcer, fatal by perforation, in which no pain was complained of prior to the acute suffering that marked this event, still it is obvious that we cannot assume the absence of so common a symptom as pain in the region of the stomach, merely because a patient has failed to speak of it at the time, or to refer to it afterwards during a brief and agonising illness. But since I have seen one or two instances in which the pain of a large ulcer has completely intermitted for several days together, I think it just possible that this symptom might be absent during the short period which sometimes includes the whole course of the disease, in cases of rapid perforation.

The character of the pain is peculiar. Rarely or never does the sufferer describe it as[1] lancinating, stabbing, or stitching. In the earlier stage of the disease, it is often little more than a feeling of weight or tightness in the epigastric region, giving the patient an impression as though the food experienced a "stoppage" here. From such a dull, continuous feeling, it gradually augments into a burning sensation, and at last into a gnawing pain, which produces a kind of sickening depression, quite distinct from the nausea often associated with it.

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[1] This character would often distinguish the pain that attends scirrhus of the stomach.

The date of its access is also characteristic. In a majority of cases it comes on from two to ten minutes ofter the deglutition of food, and remains during the hour or two corresponding to the period of gastric digestion, at the close of which act it gradually subsides and disappears. And when, as is often the case, it is accompanied by vomiting, it almost invariably ceases as soon as this act has emptied the stomach of its contents. In some instances, however, the pain immediately follows deglutition, instead of being preceded by the usual interval of a few minutes. Such a fact affords a presumption that the cardiac extremity of the stomach is the site of the lesion; a conjecture which is of course strengthened by embarrassment in the act of swallowing, suggesting its close proximity to the œsophagus. Sometimes the pain resembles that of an ordinary form of dyspepsia, in coming on half an hour, an hour, or more, after eating. Lastly, in what are generally either large lesions or protracted cases—often both—the pain loses the above character; becoming continuous during the intervals of the meals, and lasting days or even weeks without any intermission: or it even occurs chiefly on an empty stomach, and is alleviated by the ingestion of food.

The situation of the pain forms another of its characteristics. The place of its earliest appearance and greatest intensity, and to which it often remains strictly limited, corresponds to the centre of the epigastrium, or to the median line of the belly immediately below the free extremity of the ensiform cartilage. The portion of the epigastric region to which the pain is referred, forms a circular area of rarely more than two inches diameter; sometimes, indeed, a mere spot of less than half this size.

But there are exceptions to the above rule. One of these, which is not uncommon in the female, is apparent rather than real, being due to that change in the situation of the cartilages of the ribs which is effected by the compression of stays, and which materially deepens the epigastric region in the vertical direction. In other instances, the pain is behind the ensiform cartilage instead of below it; or occupies the boundary of the epigastric and umbilical regions, instead of its usual site in the middle of the former. Finally, the pain sometimes lies a little to the right or left of the median line; or extends from a point of greatest intensity here towards either hypochondrium: or, in still rarer instances, is referred chiefly to the latter situation.

In some instances, the pain in the epigastrium is associated with the feeling of violent pulsation or throbbing; in others, the same sensation is felt, independently of the paroxysm of pain, which it may even replace. It appears to be analogous to the throbbing of an abscess, and cannot be recognised by external examination.

The dorsal pain, first described by Cruveilhier, constitutes almost as important a symptom. It generally comes on a few weeks or months later than the epigastric pain, and from this time forth is almost as constant and characteristic, if not as distressing. It is a gnawing pain, which, ranging in position from the spine of the eighth or ninth dorsal, to that of the first or second lumbar vertebra, is usually "interscapular" as well as "rachidian." Like the epigastric pain, it has a fixed seat, generally remaining near the spot of its first appearance during the whole progress of the disease; and like it, also shows lateral as well as vertical deviations from its ordinary situation. But these deviations rarely remove it to a greater distance from the median line than one or two inches. Its worst attacks generally alternate with those of the epigastric pain.

How far a vertical deviation of the epigastric or spinal pain entitles us to conjecture a corresponding situation for the gastric ulcer, it is difficult to decide. Still some cases on record indicate such a connexion:—pain in the umbilical region being, for example, associated with an ulcer of the greater curvature. But with respect to horizontal deviations of these pains, there is good reason for asserting that, if well marked, they justify our inferring a similar situation for the ulcer. Out of about twenty instances of this kind which I have collected, fifteen exemplify the concurrence of pain in the left hypochondrium with an ulcer of the cardiac extremity of the stomach; and four or five illustrate the same connexion between the right hypochondrium and the pyloric extremity of the organ. My own practice has also afforded me three or four cases, in which a similar deviation led me to predict the cardiac or pyloric situation of the ulcer during life. But I have found that the more localized character of the dorsal pain makes it a better test than the comparatively diffuse epigastric pain. While I need scarcely add, that a coincidence of the two in respect to this deviation is a far stronger testimony than either unsupported can afford; and that even such an agreement requires to be confirmed by the presumptions derivable from the other characters of this symptom.

Among the latter, we may first allude to the effect of pressure in increasing the pain. This is, indeed, a very important test, being one which (to speak logically) converts what may be, for aught we know, a subjective sensitive phenomenon, into an objective one that constitutes a far more trustworthy indication of local disease. To use the expression which generally suggests itself to the sufferer, there is a "soreness" as well as a pain: the least pressure in the epigastrium is sometimes unbearable; the patient, if a female, is even content to forego the fancied advantages of her stays, rather than endure the pain which the central piece of whalebone in these ingenious aids to disease often produces. In most instances the soreness is exactly limited to the painful part of the epigastric region already specified. As it is produced by the more or less direct application of pressure to the diseased structures, it is not to be excited by pressure on the unyielding spine. But, in general, one and the same pressure on the epigastrium will excite both epigastric and spinal pain: sometimes the latter chiefly or exclusively.

Of course it is essential to use such a degree of pressure as shall not involve parts more distant than the stomach: a pressure, in short, scarcely exceeding that with which we manipulate the belly in cases of suspected peritonitis or colic. I say this, because it would otherwise be possible to make strange errors. Thus I have known cases of mere emphysema and bronchitis, in which deep epigastric pressure caused considerable distress (easily mistakeable for soreness), apparently from embarrassing the heart, which had gradually been forced down into the upper part of this region. It is not altogether superfluous to add another caution with respect to the above test. Not only must it be applied with great care and delicacy in the first examination of a supposed case of gastric ulcer, but, as a rule, we can scarcely be too reluctant to repeat it, even to verify a presumed amendment. At any rate, its effects are sometimes so injurious, that it is necessary strictly to prohibit the patient from all manipulation of the epigastric region, as well as from all pressure producible by dress (such as stays in the female) or work (as is the case with shoemakers).

Whether the pain of gastric ulcer is always increased by pressure, it seems impossible to decide. There is only one necropsy on record—and this probably not of an idiopathic ulcer—in which it is distinctly specified that pressure was altogether devoid of such an effect. But I have once or twice met with cases the symptoms of which so nearly approached those of this lesion in all other respects, that I have been obliged to suspect its presence. The varying degrees in which pressure affects different patients somewhat confirm the suspicion, that even this characteristic of the ulcer is occasionally (though very rarely) absent.

A more frequent explanation, however, of the supposed inefficacy of pressure in increasing the pain, may be found in the opposite effects producible by pressure applied in different degrees, or in different situations. Thus, a curious instance lately fell under my notice, in which pressure on the base of the ensiform cartilage relieved the patient from the sense of "stoppage," and the dull epigastric pain, which came on soon after eating. Even here, however, there was a circular area, about an inch below the apex of this cartilage, in which very moderate pressure brought on severe epigastric and spinal pain. And after the above artificial diminution of the epigastric pain, the dorsal pain soon became much more violent. Another example of the same kind has since been observed by me. In this instance, the pain was evidently relieved, not only by that pressure on the cartilages of the lower ribs which the prone attitude can produce, but even by a moderate manual pressure on the fleshy wall of the epigastrium. Even here, however, deeper or more forcible intrusion greatly increased the pain. In a less marked form such anomalies appear to be anything but infrequent: and, on the whole, justify the conjectures—that (1) pressure may either relieve or increase pain, according as it supports the periphery, or impinges upon the surface of an ulcer; and (2) that, where the lesion is so placed as to be exposed to direct mechanical interference, exceptions to the latter rule are of extreme rarity.

The effect of posture on the pain in different cases is more variable. As a rule, a severe paroxysm is relieved by the recumbent posture, no matter what may be the situation of the ulcer in the stomach. But the varieties of the recumbent posture—or, to speak technically, of *decubitus*—often have no influence whatever in increasing or diminishing the pain. But in other instances they afford a valuable confirmation to our diagnosis, and sometimes even entitle us to conjecture the exact seat of the lesion.

The facts upon which this statement is based are the following. Of cases in which the symptoms have led me to diagnose an ulcer of the stomach, about two-thirds have exhibited a marked influence of posture on the pain. During a paroxysm some were obliged to lie prone, some supine; some on the right side, some on the left, some were even obliged to sit up. In the term "obliged" to do so, I include not only those cases which were distinctly relieved by the selection of a particular attitude, but those which experienced a great increase of pain by adopting any other. In some of them, however, the painful posture has been borne for a minute or two, until the increasing severity of the pain has forced its abandonment. In like manner, the less painful attitudes have generally been adopted, to the complete exclusion of that habitual decubitus which most persons naturally assume during sleep. The remaining third have offered no peculiarity of decubitus, though in many the pain has been relieved by rest in the recumbent posture.

The fewer cases in which I have compared the effect of posture upon this symptom during life with the appearances seen after death, have afforded me more specific information to the same effect.[1] The majority have shown a close correspondence between the posture adopted and the site of the ulcer;—the prone decubitus being associated with an ulcer of the posterior surface of the stomach; the supine with one of the anterior surface; the decubitus on the right or left side, with a lesion of the cardiac or pyloric sac respectively. But, on the other hand, in some of the cases where I might most have expected such a connexion, it has been absent;—a large chronic ulcer, exclusively limited to the pyloric pouch, having been associated with no change of an habitual decubitus on the right side; and an ulcer of the posterior surface, or of the small curvature, having been relieved by even the supine variety of the recumbent attitude.

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[1] Priority in observing this interesting connexion belongs to Dr. Osborne.—*Dublin Journal of Medicine,* vol. xxvii. p. 361.

Even in these cases, however, we may probably distinguish, that the decubitus fails to guide our conjectures, rather than absolutely guides them wrong. For the fact that the habitual decubitus remains unaltered, deprives us of all grounds for any inference. Again, in the absence of specific cause to the contrary, we might well expect that the efficacy of the recumbent attitude in relieving the pain would be shared by all varieties of this posture.[1]

The partially subjective character of the pain in gastric ulcer is well illustrated by the manner in which it is often affected by sudden mental changes. Amongst these we may specially allude to the depressing passions of fear, anxiety, or anger, as capable of provoking a paroxysm which, in severity and duration, far exceeds the pain excited by distension of the stomach with food. Here, however, the situation and character of the pain generally remain unchanged.

The effect of movement upon the pain is closely connected with that of posture. As a rule, all violent bodily exertion is likely to be followed by an attack. While even moderate exercise, sustained so as to produce fatigue, generally brings about the same effect. And there can be little doubt that the relief generally afforded by the recumbent attitude is chiefly due to the perfect rest it implies. In some instances which have fallen under my notice, the movements of locomotion have given rise to a peculiar sensation of dragging in the right hypochondrium, such as induced me to suspect adhesion of the ulcerous stomach to the liver. In one of these instances I have since verified this conjecture by a necropsy.

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[1] The presence of such a correspondence in certain cases, and its absence in others, naturally remind one of what may be called objective and subjective sensations in the normal action of nerves, and suggest the analogous distinction of objective and subjective pains in their abnormal states of activity. But such a view fades before any philosophical inquiry. Even in the commonest forms of irritation of a nerve, most of the minute mechanical conditions remain unknown to us: and yet, until we can specify and determine these, we cannot assert that a given pain is not so far objective, as that it results from a local lesion of the nerve. In like manner a careful consideration would probably conduct us to the propositions;—that all pain is subjective; that nothing but an elaborate organization at its periphery and centre enables any nerve to give an objective sensation; and that, even then, its objectivity is, strictly speaking, but very partial and imperfect.

Here I may perhaps mention another effect of adhesion, which well illustrates the accuracy of an old observation respecting the symptomatology of the liver. In two or three cases, the adhesive inflammation uniting the ulcerous stomach to the surface of the liver has been accompanied by that pain in the right shoulder which has long been regarded as characteristic of hepatic inflammation.[1]

It has been suggested that the pain of gastric ulcer is connected chiefly with the extension of the disease to the peritoneum, or with its location at the cardiac or pyloric orifices. That either of these peculiarities would, on the whole, tend to increase the pain, there is good reason to suppose. But that intense and continuous pain is quite independent of them both, I can affirm from a large number of necropsies.

The pain is also affected in a special manner by food. As already mentioned, its paroxysms generally have a close correspondence with that period of gastric digestion during which the organ is distended with food. It is increased by the ingestion of hard or indigestible substances; and it is mitigated by a pulpy and bland diet. There are also many articles of food which have special irritating effects. Among liquids, few are more generally unbearable than tea or beer. Finally, hot substances are usually productive of great pain.

But exceptions to all these rules are occasionally seen. The pain is sometimes unconnected with the ingestion of food; sometimes relieved by it. And I have known even brandy, or warm water, taken by a patient with relief. While careful inquiry has satisfied me that beer is sometimes (especially in the aged) of no injury to the stomach, and of advantage to the organism generally.

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[1] This sympathetic pain belongs, I believe, chiefly to superficial inflammation of the liver; and is mediated by the nerves of the diaphragm and wall of the belly.

Lastly, in the young female, the pain of gastric ulcer is often affected by the access of menstruation. Sometimes the pain thus provoked seems quite distinct from that previously present, being abdominal and lumbar (instead of epigastric and dorsal) in its site; unaffected by pressure; unconnected with vomiting; in short, evidently dysmenorrhœal:—a conclusion which is confirmed by the fact, that it recurs at the menstrual periods, long after every symptom of the gastric malady has disappeared. But in other instances the access of the menstrual flux has a specific (and sometimes habitual) influence in provoking and increasing the ordinary pain of gastric ulcer, with all its usual concomitants (tenderness to pressure, vomiting, peculiar decubitus, &c.) The nature of the relation between the pain and the menstrual epoch is shown by its mode of access;—commencing, as it usually does, from twenty-four to forty-eight hours before the appearance of the flux: when it generally begins to subside, to disappear about twenty-four to thirty-six hours after the commencement of free sanguineous discharge. It is thus connected more intimately with the menstrual *molimen*—with that disturbed state of abdominal innervation which precedes the flux—than with the flux itself.

The *vomiting*, which generally forms the next symptom in the history of gastric ulcer, is far from exhibiting characters equally specific with those of the pain. It usually occurs when the paroxysm of pain has reached its greatest height; and is therefore rarely of a violent character, the distension of the stomach which prevails at this time sufficing to render it an easy and painless kind of vomiting. Once begun, it seems rarely to end without emptying the stomach of its alimentary contents:—an act of expulsion which usually affords complete relief to the pain, but sometimes leaves a slight burning sensation, that only disappears after a variable interval of time.

The chief varieties of the vomiting relate to the following details. Firstly, as regards the date of the malady marked by its access, though generally preceded by the characteristic pain during several weeks, it occasionally comes on almost as soon as this symptom. As respects the substances it expels, these vary chiefly with the precise date of the act. Soon after the ingestion of food they are of course alimentary; at a later period they have an acid character, often occasioning an intensely sour taste to the patient himself; and still later, are sometimes mixed with bile. Lastly, in those rarer instances in which the act of vomiting comes on quite independently of the ingestion of food—for example, shortly after rising from a night's sleep—it expels a glairy alkaline fluid, consisting chiefly of the saliva swallowed before the attack. Here the vomiting, often periodic, is sometimes connected with habitual drunkenness; especially with the collapse which follows a debauch.

As regards the proportion of cases in which gastric ulcer is attended by this symptom, I believe that it is rarely absent from the whole course of the malady, except in the rapidly perforating ulcer of the young female. Sometimes, however, it is so easy in character, and expels so little of the gastric contents, as to merge into regurgitation :—a symptom which often ushers in vomiting, and occasionally remains as its sole representative throughout the whole course of the malady. In some cases it has been represented by mere nausea in ulcers which have remained active for years. In others it has been limited to a single attack, or to the very close of the disease. Evidence to this effect may also be found in the result of a strict regimen in alleviating vomiting; which in many cases only comes on after a full meal, and is at once suppressed by reducing the food to a minimum of the blandest alimentary substances. Among other circumstances favouring the access of vomiting, there is but one which seems to have any close and constant relation to its frequency and intensity ;—namely, the size of the gastric ulcer. But this character is often connected with a long duration of the lesion, and with its adhesion to neighbouring organs: two circumstances which may independently favour the occurrence of vomiting.

The danger of this symptom it is difficult to exaggerate, though easy to explain. By expelling the food shortly after its reception into the stomach, it starves the patient; with a rapidity determined chiefly by the quickness of its access, and the completeness with which it empties the organ. And in addition to the effects of inanition, it adds the fatigue implied by violent and abnormal action of the nervous and muscular systems. (Compare p. 52).

Hæmorrhage is the next symptom of gastric ulcer. Since the process of ulceration itself implies a solution of continuity in the coats of some of the vessels of the stomach, nothing short of a simultaneous obliteration of these tubes can prevent some effusion of their contents. And hence it is not very surprising to find that the myriads of vessels concerned in every ulceration are rarely occluded with that quickness, precision, and universality, requisite to prevent all hæmorrhage from their interior.

How far the hæmorrhage which occurs in ulcer of the stomach may be attributed to mere congestion, it is impossible to determine. But from analogy, we may presume that the same degree of congestion which often attends ulceration elsewhere, might constitute an equally efficient cause of bleeding in the stomach. Still, as the hæmorrhage almost invariably occurs soon after a meal, and is often distinctly traceable to the ingestion of an unusual quantity of food, we may suspect that the influence of such an inflammatory congestion is far surpassed by that of the afflux of blood which attends the act of gastric digestion; as well as by the mechanical disturbance which distension of the stomach necessarily inflicts on the diseased vessels of the ulcer itself.

There are no data for determining the frequency of those scanty hæmorrhages which are poured out, in the earliest stages of the ulcerative process, from the minute vessels of the mucous membrane, and of its sub-mucous areolar tissue.[1] But it is certain that they occur in a large majority of cases, in many of which no symptom reveals them. For since a small quantity of blood does not excite vomiting, it depends entirely upon a casual coincidence of these two symptoms—hæmorrhage and vomiting—whether the former is revealed by the latter. And unless attention be particularly directed to the stools, a moderate quantity of blood may also leave the intestinal canal by this channel, without ever being detected.

In cases of this kind, the blood exhibits the changes which ordinarily attend its exposure to the action of the fluids of the stomach and intestines; not only becoming mingled with the various ingesta and secretions which may chance to be present, but gradually undergoing a process which has the effect of greatly modifying its colour and consistence. Wherever the extravasated blood is sufficiently exposed to this action, it acquires a dark, grumous, or even black colour, and a peculiar tarry or almost pultaceous consistence. A small quantity of blood thus altered by digestion sometimes even simulates the colour and appearance of inspissated bile.

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[1] Compare the remarks on the sources of such hæmorrhage in a subsequent part of this Lecture.

Hence the following precautions are applicable to this symptom of gastric ulcer. We cannot presume it has been absent because it has not been noticed by the patient. Our inquiries must be directed not only to the matters vomited, but also to the stools. As regards the former, we must question the patient, not only as to what he may have recognised as blood, but as to the characters of all the substances he has vomited. And the matters habitually rejected from the stomach should be submitted to a strict and repeated microscopic examination; care being taken to select such specimens as are free from all admixture of food—at any rate of animal food containing blood. Precautions of this kind will often show that a comparatively clear fluid deposits a sediment containing blood corpuscles in considerable quantity; and perhaps ranges, in other specimens from the same patient, through a brownish ropy mucus, to a grumous fluid having the ordinary "coffee-grounds" appearance of blood thus altered by digestion. A similar examination will sometimes be useful in the case of the blackened fæcal evacuations to which gastric hæmorrhage gives rise. Dilution with water will generally distinguish inspissated bile. But if not, the microscope will at once set this question also at rest.[1]

The proportionate frequency of those larger hæmorrhages which occur when the vessels external to the stomach become involved in the ulceration, is just as uncertain. But my own experience would suggest that they occur in not more than one-third of the gastric ulcers which come before us in ordinary practice.

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[1] The ingestion of the salts of iron is a source of error that may of course be easily detected; though I have known the inky vomiting which has accidentally followed the administration of this drug immediately after tea, excite considerable alarm in the mind of a patient and his medical attendant.

The symptoms of such hæmorrhages illustrate and confirm the proposition implied above;—namely, that the blood poured out from a gastric ulcer scarcely exerts any specific action as an emetic or a purgative, but excites vomiting or diarrhœa chiefly by its quantity; in other words, by the mechanical stimulus which its distension of the stomach or bowel supplies. Soon after a meal, the patient begins to experience an unusual fulness and weight in the region of the stomach; attended (sometimes even preceded) by feelings of syncope. Nausea rapidly supervenes, and ends in the vomiting of a large quantity of blood; which may either be partially coagulated, or, if rapidly effused and rejected, may retain a colour and fluidity that testify to its arterial source. In other cases (and I am disposed to conjecture, chiefly in those rarer instances where the hæmorrhages, besides being smaller, occur independently of a meal), the blood is effused in considerable quantity without exciting any vomiting whatever; and is passed at once through the pylorus into the intestine, which it leaves more[1] or less rapidly with the stools. Lastly, in very exceptional cases, the rapidity of the hæmorrhage is so great, that it distends the stomach and more or less of the intestine with a single gush; and the patient faints and dies before any expulsive act can diminish the enormous clot which the necropsy reveals as the cause of his sudden decease.

The *state of the bowels* in this malady seems unconnected with any special character of the lesion. Constipation is, however, the rule; a fact which appears to have a twofold cause. Firstly, the ulcer itself generally opposes the reception, and ensures the expulsion, of ingesta; and thus deprives the intestinal canal of those contents which require, and provoke, the act of defæcation. Secondly, there is a definite[2] quiescence, or stagnation (so to speak) of the intestinal walls—an arrest or exhaustion of peristalsis—which is produced by peritonitis, by vomiting, and probably also by intensely painful affections of the stomach: and which is therefore especially marked in many instances of gastric ulcer where all these circumstances concur. Diarrhœa is so much the exception, that its frequency seems scarcely much greater than might be expected, supposing it quite independent of the lesion. But, as already intimated, copious hæmorrhage generally gives rise to looseness of the bowels. A significant contrast to this fact is afforded by ulcers situated in the first portion of the duodenum (or in the immediate neighbourhood of the stomach), which give rise to diarrhœa with much greater frequency than does gastric ulcer. There can be little doubt that this difference is due to a simple law of the peristalsis of the alimentary canal: a law in accordance with which the movements of the most distant parts of the intestine are connected together, while they are comparatively isolated from those of the stomach. Hence copious and frequent diarrhœa, especially if attended with a moderate hæmorrhage of scarcely blackened blood, points rather to the intestine than to the stomach: and should direct inquiry to the precise seat of the abdominal and dorsal pain, as well as to the symptoms of those maladies (phthisis, typhoid fever, &c.) which are known to be often associated with ulceration of the ileum and cæcum.

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[1] Indirectly, it may doubtless provoke diarrhœa, by undergoing decomposition in the bowels.

[2] Compare p. 66, *et seq.*

Amenorrhœa is so frequent a symptom of gastric ulcer in the female, as to require a special consideration.

The exact frequency with which the presence of this symptom coincides with the existence of the ulcer cannot be estimated. But on the whole, regular menstruation seems far more common than is generally supposed: a fact which is quite in consonance with what will be hereafter stated respecting the number of males, and of females either past the menstrual epoch or not arrived at puberty, in whom the lesion has been detected by careful necropsy.

Further, even in the female during the menstrual epoch of life, this symptom appears to be associated with different groups of the lesion, in very different degrees.

It is in the chronic ulcer of middle-aged women that the catamenia are least affected. Many of the cases in which the ulcer has lasted for ten or fifteen years, are recorded to have menstruated regularly; some even profusely. Indeed, in some the malady has lasted throughout the whole menstrual epoch of life, without exercising any appreciable influence on this function.

The coincidence of amenorrhœa with copious hæmorrhage from the ulcer is certainly more frequent. But the two symptoms generally have a very natural and obvious relation. The amenorrhœa follows the hæmorrhage; and is caused by it, just as it would be by any other hæmorrhage, or by that drain of nutritious fluids which pregnancy or lactation might imply. In other instances, the amenorrhœa precedes the hæmorrhage. But since hæmorrhage is not more frequent in these cases than in cases of chronic ulcer in general, there is no ground for asserting the efficiency of suppressed menstruation as an independent cause of the bleeding. In like manner, there is rarely any connexion between the date of the hæmorrhage and the menstrual period. And finally, whatever has been written respecting the liability of the gastric ulcer to give rise to a periodical hæmorrhage which forms a vicarious menstruation, I do not know a single well-authenticated instance of this kind on record.[1]

There is, indeed, but one group of gastric ulcers with which amenorrhœa seems to have any intimate relation: namely, the perforating ulcers of the young female.

The majority of these cases exhibit scantiness or absence of the menses as one of the most prominent features of their history. In many of them the amenorrhœa is accompanied with a state of pallor and anæmia, resembling chlorosis. Some, however, menstruate regularly and copiously: and a few profusely. While many are instances of delay in the appearance of the menses, rather than of their suppression or interruption. And in consonance with this, it may be pointed out, that though the age[2] of many of these female subjects of perforation closely approaches the epoch of puberty, and the year or two immediately following, yet it does not exhibit those fluctuations above and below the average which would assign it exactly to the access of this state.

Still, the coincidence between amenorrhœa and the perforating ulcer is an unquestionable fact. And the first question concerning this fact suggests itself in the form of an alternative.

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[1] Compare the author, *op. cit.*, p. 85.

[2] See p. 145.

Does the amenorrhœa cause the ulcer, or the ulcer the amenorrhœa?

The first of these two questions I think we must answer in the negative;—not only because the ulcer is present in the male sex, and in the neuter monster,[1] at the same age, as well as in the female at all other ages; but because, even in the female at this epoch of life, the exceptions to the presence of the supposed cause are too numerous to be compatible with such a causation. Indeed, to the various cases of regular menstruation thus alluded to, we might plausibly add a large proportion of those in which menstruation had delayed its appearance, as well as all those in which puberty was absent. For many of the former would scarcely be instances of mere amenorrhœa, just as all the latter are certainly disqualified for this epithet.

In favour of an affirmative answer to the second question (or of the view that it is the ulcer which causes the amenorrhœa), we may point out:—that, in most of these cases, the dyspeptic symptoms indicative of the establishment of the lesion have themselves preceded the deficiency or cessation of the menses; and that such an explanation, just as it would receive no contradiction from the mere age of these cases, so it would find a parallel in the case of other grave constitutional disorders which, no pathologist would doubt to be the cause of the amenorrhœa frequently associated with them. A good illustration of this statement may be found in the tuberculous cachexia, which often selects this epoch of female life as the period of its fatal attack, and is associated with symptoms of chlorosis and amenorrhœa.

A careful observation of the details of the so-called "chlorotic" state which accompanies the amenorrhœa of these gastric ulcers, affords some confirmation of the above view. Not only is the cachexia present by no means identical with true chlorosis, but the differences of the two states are essential. The cachexia which attends the ulcer is, even when best marked, devoid of the characteristic symptoms of severe chlorosis. The pallor, however extreme, offers no trace of that greenish hue which the very name of chlorosis (χλωρὸς, *green*) connotes. The dyspnœa on exertion, and the soft bellows-sound, are much less distinct.

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[1] Compare the author, *op. cit.*, p. 32.

And lastly, there is little or no œdema of the subcutaneous areolar tissue.

As the age of these subjects of gastric ulcer advances, it is not uncommon for the amenorrhœa to cease, the other symptoms of the lesion remaining unaffected. In rarer instances, the so-called chlorosis also diminishes and disappears. But the most satisfactory proof that the amenorrhœa is not in any sense the cause of the chlorotic symptoms, is afforded by some still rarer (though authentic) examples, in which the ulcer has been attended by a marked degree of this cachexia, without any interruption whatever to regular and copious menstruation.

But it is obvious that the above view does not explain the connexion between amenorrhœa and perforation; much less the fact, that the ulcer most affects the menstrual function soon after the age of puberty. Nor do I think that sufficient materials for such explanations are at present accessible. It would, however, be easy to suggest, that a periodic hæmorrhage like the menstrual flux may well be more easily affected during the struggles of the constitution to establish and maintain it, than when the organism has become accustomed to its recurrence, or strengthened, so as to be more indifferent to the loss of blood it implies.

But this epoch of female life seems to influence, not merely the symptoms, but also the characters of the gastric ulcer. That its great liability to perforation is not due to any supposed acceleration of the ulcerative process by the vigour and activity of youth, is shown by the fact, that the increased tendency to perforation at this age is limited to the female. (Compare p. 145.) Indeed, all the peculiarities of the symptoms and appearances seen in this group of ulcers concur to explain their tendency to perforation as being due, not to any special activity or quickness of the ulcerative process, but rather to the absence of that inflammatory reaction by which its destructive advance is often checked, and its worst effects warded off.

The *cachexia* generally associated with the ulcer at other ages of life appears to have precisely the same import as the chlorotic condition which represents it in the young female.

Like the latter, it seems to be essentially, not so much a symptom, as a congeries of symptoms: or rather a state which expresses the injury inflicted on the organism by a variety of causes. The wearing effect of long and frequent paroxysms of pain, the fatigue and inanition implied by constant vomiting, the drain of frequent or copious hæmorrhage, the loss of digestive power involved in the destruction of the stomach, and finally, the mere age of the patient—are causes of this kind, each of which often shares in producing the cachexia that is present.

As already intimated, it is probable that this cachexia (which is best marked in ulcers of long standing, and therefore in middle-aged or elderly people), corresponds to the chlorotic symptoms and the amenorrhœa above noticed as generally associated with gastric ulcer in the young female. And my own experience entitles me to presume that it is rarely absent; that (contrary to what is often stated on this point) a person suffering from gastric ulcer scarcely ever offers an appearance such as an observant practitioner would mistake for that of a person in health. Nay, more, I may add, this cachexia gives the disease so peculiar a physiognomy, that I have sometimes been forewarned of its presence by the mere sight of the patient's features in a crowded Hospital out-patient room. With some characters that would often leave us in complete uncertainty as to whether the cachectic aspect was due to ordinary chlorosis, to tuberculosis, or (in later life) to the cancerous diathesis, the sharp lines which severe and frequent pain, with partial starvation, have worn in a patient's face, sometimes afford what is thus almost a characteristic sign of gastric ulcer. At any rate, this peculiar expression of countenance is, on the whole, a surer indication than mere anæmia, emaciation, or exhaustion can afford.

The *perforation* which sometimes occurs in the course of gastric ulcer is notified by symptoms so intense and characteristic, as to require but a very brief description. After more or less distinct indications of the ulcer have existed during a variable period, the patient is suddenly attacked by a violent pain, which begins in the epigastrium, and rapidly spreads over the belly. Its diffusion is accompanied by the appearance of all the ordinary symptoms of peritonitis; the wall of the belly becomes extremely tender to pressure; the patient assumes an attitude which relaxes the muscles of this part; there is a cessation of the usual respiratory movement here—a stagnation soon followed by great tumefaction of the abdomen and tympanitic distension of the bowels. If a strict physical examination of the belly be insisted on, the peritoneal cavity will be found to contain fluid: usually those contents of the stomach which have streamed through the aperture in its coats, increased by the subsequent addition of an inflammatory effusion. The continuance of these symptoms generally destroys the life of the sufferer in from twenty-four to thirty-six hours; but death is often preceded by a period of comparatively painless collapse.

Rarely does the train of symptoms that follows perforation offer any marked deviation from the above type.

In many instances, however, a remarkable paroxysm of pain precedes the occurrence of perforation. This intense pain—the duration of which varies from a few minutes to several hours—is, I believe, generally due to a leakage of the gastric contents through that thin film of rotten tissue, to which, at this period, the coats of the stomach are reduced. In consonance with such an explanation, a more chronic pain of similar character has sometimes been found associated with a complete matting together of the stomach and all the neighbouring viscera by a large quantity of lymph, without any visible perforation of the coats of the stomach, or any escape of its contents.

The symptoms of those various modifications[1] of the process of perforation which will be noticed in treating of the pathology of the gastric ulcer, require no special description here. Partial perforation, allowing of a subsequent repetition (or rather extension) of the accident, or leading to abscess, is distinguished by symptoms which, though scarcely quite alike in any two instances, offer the general features of being more local, more chronic, and less intense, than those of ordinary perforation fatal by general peritonitis.

There are other circumstances attending the accident which might almost be enumerated among its symptoms. Thus it almost always occurs after a full meal; and is often directly traceable to mechanical violence, such as coughing, sneezing, concussion or constriction of the belly. The sensations of the patient frequently verify the nature of the accident, by distinctly appreciating that something has given way in the belly; and has thus caused a gush of fluid which has instantly provoked an agonizing pain in the same part. Lastly, the presence of a communication between the cavity of the stomach and that of the peritoneum is sometimes indicated by the repeated recurrence of intense pain after the deglutition of food or medicine.

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[1] Compare p. 148.

Dilatation or enlargement of the stomach, as a result of the constriction produced by the cicatrix of a gastric ulcer, constitutes a variety of the malady which of course brings with it a special train of symptoms. Into these, however, I shall not now enter, but shall content myself with pointing out that their connexion with the ulcer is both infrequent[1] and indirect; while they are present in instances of dilatation from so many other causes, that nothing short of a history revealing those general symptoms of the ulcer already described would entitle us to refer any particular case to this origin.

The above varieties in each of the symptoms of gastric ulcer suggest, on merely arithmetical grounds, the infinite modifications which necessarily result from their combination. Of these it is scarcely an exaggeration to say, that they make each case of ulcer of the stomach unlike every other. And they especially raise two questions respecting the diagnosis of the malady. (1) What is the minimum of evidence which will justify us in affirming the existence of an ulcer of the stomach during life? (2) What are the diseases with which it is most likely to be confounded?

A specific answer to the first of these questions it is impossible to give. But I am inclined to think that nothing less than a concurrence of all the chief symptoms, as already described, entitles us to pronounce a decided opinion. In other words, unless the pain possess the characters attributed to it, and is accompanied by an equally characteristic vomiting; and unless there be evidence of considerable or repeated hæmorrhage in the course of the malady; there is no sufficient ground for affirming the existence of gastric ulcer. The drift of these symptoms may be thus summed up. The date, duration, and frequency of the pain chiefly indicate some important lesion of the mucous membrane of the stomach. The vomiting adds, that this lesion implies great irritation of the nervous centres connected with the organ. And it is reserved for the hæmorrhage to show, whether the disease is one which involves an absolute breach of continuity in the structure of the stomach.

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[1] Infrequent, as not occurring much oftener than once in 200 cases; indirect, as implying in most instances a previous cessation of the ulcerative process, or healing of the ulcer.

But an absolute enforcement of this rule of diagnosis would lead us to overlook many cases of gastric ulcer; and thus occasion grievous errors in practice. In point of fact, even when any definite opinion is impossible, we may often suspect that the symptoms are due to this lesion.

Thus, as already mentioned, the pain is subject to considerable variety in many of its features. As might be expected, a moderate hæmorrhage sometimes escapes the notice of both physician and patient. And even where the latter habitually inspects whatever is discharged by stool or vomit, or the physician calls in the aid of the microscope to an examination of any suspicious egesta, the irregularity of its occurrence may baffle all attempts to verify it for months together. In like manner, the vomiting seems to be sometimes (though much less frequently) absent from the history of the malady during a great part of its course; or merges into a trifling regurgitation after meals, such as we hardly dare consider its representative.

Indeed, a careful consideration of the details already adduced affords a complete explanation of many such anomalous cases of ulcer of the stomach. For the slow succession of the symptoms in a majority of cases almost implies their absence in a minority. The delay of this or that particular symptom not only deprives us of the (multiplied and not merely added) probability it contributes to our diagnosis, but merges two or more stages of the malady into one, or even reverses their order of sequence. And as the lesion itself may be fatal at any period of its progress, it is obvious that the casual delay of any symptom—perhaps for a period not greatly exceeding that of the interval which generally precedes its being added to those previously present—is sometimes scarcely tantamount to its absence, even though, as a matter of mere narrative, it has never been noticed.

Obscure and uncertain cases of this kind show the importance of a thorough knowledge of the characters of the disease in its more chronic and typical forms. The pathology of the lesion in general has to supply any casual deficiencies in the physiognomy (so to speak) of the particular instance. Above all, we must remember that it is our first duty to be useful; and that suspicions which fall far short of a definite diagnosis, may yet be sufficiently important to dictate the whole plan of treatment. Suppose, for example, that we are consulted by a patient for protracted or severe dyspepsia, which has seriously affected the general health, and is associated with pain and tenderness in the epigastrium, and pain in the interscapular region, increased or provoked by the ingestion of food. If, on further inquiry, it turns out that this pain is especially called forth by proteinous substances, or by hot liquids; and that it is affected as above described, by movement, rest, and posture; there can be little objection to our keeping steadily before us the possibility of a gastric ulcer. Such a suspicion, it is true, guides us to a specific course of treatment: but that treatment involves neither pain nor danger, and a diet scarcely more stringent than what many a dyspeptic would gladly submit to for the removal of his distressing symptoms. Indeed, it is hardly too much to say that, by treating these cases as ulcer of the stomach, we may often cure what we cannot diagnose; and—to venture upon a paradox—may thus witness such a triumph of the Art over the Science of Medicine, as well illustrates the invaluable (but yet subordinate) relation of clinical and pathological research to the one great object towards which all our knowledge should tend—the successful treatment of disease.

These remarks will especially apply to such symptoms when they occur, in connexion with amenorrhœa, in females who have lately arrived at puberty. Here the absence of hæmorrhage, and the little attention often given to mere dyspeptic symptoms, sometimes obscure the diagnosis of cases, in which a careful inquiry into the history of the malady, and a sedulous examination of the epigastric region, afford only too much reason for supposing that the patient is in imminent danger of death by perforation of the stomach.

The second question—"With what diseases is gastric ulcer most likely to be confounded?"—could only be fully answered by details of cases such as cannot here be adduced. Dyspepsia, chronic inflammation, and cancer of the stomach; disease of the duodenum; gall-stones; abdominal aneurisms; enteric tuberculosis; and a variety of diseases too numerous to mention;—all present various degrees of similarity to gastric ulcer. And the variable symptoms of this lesion often render such similarity much more suggestive of error than is the case in the maladies of many other organs.

The above observations render it unnecessary to dilate upon the circumstances which generally distinguish dyspepsia from gastric ulcer. In most instances, there is little difficulty in deciding which of the two maladies is present. But in some cases the distinction is by no means easy. And of all the Protean forms which dyspepsia may assume, that called the "morbid sensibility" of the stomach is the one which is most likely to include cases of ulcer; or, in other words, if really independent of this lesion, is most likely to be mistaken for it.[1]

Of the other diseases just enumerated, there is none in which the resemblance to ulcer is so close, and a definite opinion so important, as in the case of cancer of the stomach. Hence we may enumerate (though we cannot now discuss) the chief considerations on which their differential diagnosis would generally depend. The cancerous disease especially affects the epochs of middle and advancing life. Its symptoms rarely date from more than twelve or eighteen months prior to the death of the patient. It is associated with the cancerous cachexia: often with cancerous disease of other organs. In most cases it forms a hard but moveable tumour in the epigastrium. Its pain generally has a lancinating character; and a time of appearance belonging rather to the later stage of gastric digestion than to the few minutes which succeed deglutition. Its hæmorrhage is more scanty; and is, on the whole, later in the history of the malady. Its vomiting is also generally late; rarely of many months' continuance; and expels what the microscope will often show to be a cancerous cell-growth. But unless unusually distinct, scarcely one of these characters possesses much independent value. For the gastric ulcer is frequent in middle and advancing life. It may destroy life in a few days or weeks. It is often associated with cachexia; which, again, is sometimes quite undistinguishable from the cachexia of cancer. It is not unfrequently accompanied by pulmonary disease, such as can tolerably simulate secondary cancer of the lungs. The lymph by which an ulcer adheres to the liver or to other viscera may give rise to a tumour, which can be felt through the wall of the belly. Its pain may affect a lancinating character, and be deferred until some time after meals. Its hæmorrhage may have the moderate amount, and the "coffee-grounds" appearance, ordinarily seen in that of cancer. And unlikely as it may seem that many of these separate and infrequent contingencies should combine to obscure the diagnosis of any single case, such instances really do occur. Once or twice I have myself met with cases of this kind, in which there was nothing to justify any definite diagnosis between the two diseases; and in which the moderately large ulceration detected months before death has offered no symptoms during the whole time which could warrant its being definitely diagnosed as malignant or the reverse.

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[1] See the remarks on this subject in Lecture VI.

PATHOLOGY.

It has already been stated that the preceding description of the symptoms of gastric ulcer is derived from a large number of cases, directly or indirectly authenticated by necropsy. The changes which constitute the progress of the disease have also been deduced from the examination of a series of specimens of its lesion, in successive stages of its existence. We have now to add some details respecting its appearances in the dead subject; details which, besides interpreting the symptoms and (as we shall hereafter find) guiding the treatment of this malady, suggest various deductions of great interest in reference to its origin and causation.

As respects the *frequency* of ulcer of the stomach, the lesion appears to be detected in from 2 to 13 per cent.—on an average, 5 per cent.—of persons dying from all causes: the higher proportions probably corresponding with habits of spirit-drinking, on the one hand, or with a high average age of the persons whose bodies were submitted to such systematic necropsy, on the other.

Sex.—As long believed, the ulcer is more frequent in the female than in the male sex, the proportions being nearly as 2 to 1 respectively.

Age.—As respects age, it would appear from a comparison of the number of ulcers detected at different ages with the proportionate numbers of persons of these ages living in this country, that the liability of an individual to become the subject of gastric ulcer gradually rises, from what is nearly a *zero* at the age of ten, to a high rate, which it maintains through the period of middle life; at the end of which period it again ascends, to reach its maximum at the extreme age of ninety. We may therefore conclude that ulcer of the stomach is specially, though not exclusively, a disease of middle and advancing life.

The *situation* of the ulcer may be summed up by the statement, that the lesion occupies the various parts of the stomach in about the following frequency. In 43 cases per cent., the posterior surface; in 27, the lesser curvature; in 16, the pyloric extremity; in 6, both the anterior and posterior surface, often at opposite places; in 5, the anterior surface only; in 2, its greater curvature; in 2, the cardiac pouch. Thus about 86 ulcers in every 100 occupy the posterior surface, the lesser curvature, and the pyloric sac:—parts of the stomach which together form a segment of less than one half of the total superficies of the organ. Hence we may estimate that any part of this continuous (but irregular) segment of the stomach is, on an average, about five times more liable to the lesion, than the remaining segment formed by the cardiac sac, the anterior surface, and the greater curvature.

In *size*, the ulcer is rarely much smaller than a fourpenny-piece, or larger than a crown-piece. But no precise limits can be assigned it. Thus an ulcer not larger than a pea may exhibit all the characters of this lesion, and cause death by hæmatamesis, or perforation. While, conversely, an ulcer may gradually extend to a diameter of five or six inches; in other words, to $\frac{1}{8}$th or $\frac{1}{9}$th of the total mucous surface of the stomach.

The *shape* of the ulcer is usually circular or oval. But it is often oblong, with a direction either parallel or transverse to the axis of the stomach; and in rare instances it forms a zone around the pyloric valve, or the pyloric sac. Some of these irregular shapes are evidently due to the fusion of two or more ulcers into one, by the progressive enlargement of their adjacent margins.

As regards the *number* of ulcers, two or more are present in 1 out of every 5 cases, or about 21 per cent. Of 97 such plural cases (corresponding to 463 instances of ulcer): in 57, there were two ulcers; in 16, three; and of the remaining 24, in which "several" ulcers were present, 3 cases offered four, and 2 cases five ulcers each; while in 4 there is reason to suppose even this number was exceeded.

Margin.—The appearances of the tissues in and around the ulcer are subject to just as much variety as its size and shape. In some instances, there is little or no evidence of inflammation in the neighbourhood of the lesion; which consists of a mere removal of the mucous membrane over a circular space, that forms a shallow but level pit, with a sharp, smooth vertical edge, as though it had been punched out. In other cases, which appear to form the majority, the mucous membrane that constitutes the immediate margin of the ulcer is swollen, so as to be raised somewhat above the level of the adjacent mucous surface. And a microscopic examination shows that this thickening, which is accompanied by induration, depends upon an exsudation of lymph into the areolar tissue beneath the mucous membrane, as well as into the matrix of the latter texture itself:—constituting a slight but appreciable inflammatory reaction, which, in respect of its nature, is closely akin to that adhesive inflammation of the peritoneum hereafter mentioned.

In many instances, indeed, the swelling and induration around the ulcer far exceed that just described: and convert the mucous membrane, for the distance of half an inch, an inch, or more, into a thick brawny mass, which has sometimes been mistaken for cancer. Rarely, however, will a careful examination leave us in any doubt as to its nature. Even when best marked, the total increase of thickness in the parietes of the stomach is but moderate. The exsudation which causes this increase of thickness is often confined to the mucous membrane and the areolar tissue immediately beneath it: and consists of fibres, in which it is usually very difficult to find even moderate quantities of the cell-growth from which they are developed. Hence the new substance has neither the structure nor the situation of the cancerous deposit (see Lecture IV). The mucous membrane, itself, however thickened, remains in what is essentially a healthy state. Indeed, in many such instances it seems little more than hypertrophied, in the strictest sense of this term. And, lastly, the history of the lesion would generally afford sufficient grounds for a decision, even prior to an inspection of its appearances.

The latter allusion suggests an explanation of the maximum, minimum, and medium of that inflammatory reaction indicated in the above remarks. As might be expected, the simple, punched-out ulcer is usually either a small or recent lesion, on the one hand, or is associated with a weakly and cachectic (in the female, often chlorotic) state of constitution, on the other. While the maximum of thickening is generally found in connexion with the same circumstances which favour the occurrence of adhesive inflammation on the exterior of the stomach:—and among these, especially with a large size and long duration of the ulcer. But it frequently occurs in subjects of the age of twenty or twenty-five. Still this fact does not qualify the preceding statement as to the usual duration and diameter of the indurated ulcer, but seems merely to express the degree in which the process of reactive inflammation is sometimes furthered by the vigour of youth.

Such varieties in the size, shape, appearances, and numbers of the lesion, show in what a restricted sense we must adopt the nomenclature by which it is generally known in medical treatises. It is generally called *the ulcer*—often the *simple*, or *chronic*, or *perforating ulcer*, of the stomach. Now, as regards the word *ulcer*, an important exception may be taken to its use. For the comparison of any large number of specimens would conclusively show, that there is no specific pathological distinction between "ulcer" and "ulceration" of the stomach; and that all the distinctive characters which the most minute description could assign to either, merge into those of the other by infinite gradations. It is true that numerous or large ulcerations produced by a rapid process of destruction, are rarely or never accompanied by those appearances which imply even a moderate duration; that they are devoid of raised edges or a hard margin, and do not penetrate the larger vessels, or even the total parietes, of the stomach. But, practically speaking, all this amounts only to the statement of a very obvious fact:—namely, that such lesions destroy so large a fraction of an organ essential to nutrition and life, that the unhappy subject of them either dies before they have time to offer appearances of reaction, or is too prostrate to be amenable to the inflammatory process. While each of the other terms emphasised above will be found to illustrate the rule of "*lucus a non lucendo.*" Though the lesion is called *the* ulcer, it is neither single nor definite in its nature and origin; and is often present in the plural number. Though it is called the *simple* ulcer, its characters are generally a compound of two processes of absorption and reaction, the latter of which certain instances show to be quite independent of the former. Though it is called the *chronic* ulcer, its progress is sometimes so rapid, as to penetrate the stomach, and destroy life, in a few days. And, finally, though it is called the *perforating* ulcer, in about seven out of every eight cases it does not perforate.

The *base* of the ulcer, so long as it is formed by the tissues of the stomach itself, presents appearances similar to those of its margin. Its usually firm and hard consistence is derived, partly from the density of the areolar and muscular tissues originally present, partly from an increase of their cohesion, due to that infiltration of fluid, and exudation of lymph, which inflammation generally brings about. In other (and by no means infrequent) cases, the progress of ulceration, apart from any such reaction, is betrayed by the soft, flocculent, or even gelatinous consistence of the floor of the ulcer; where we sometimes find flakes of dead tissue, the size of which almost entitles us to regard them as sloughs.[1]

But since the ulcer, beginning in the mucous membrane, gradually extends through the coats of the stomach, so as to increase its depth, as well as its surface, its base and margin are necessarily subject to continual change. Still the mode by which it penetrates the various tissues of the stomach is so characteristic, that there is little alteration in the shape of the ulcer, so long as it does not pass beyond them.

The whole depth of the ulcer forms a cone, of which the base is at the free internal surface of the stomach, while the apex points towards the peritoneum. The smooth, sharp, vertical edge which forms the lateral boundary of the ulcer as it passes through the mucous membrane, is exchanged for a smaller and less regular circle where it reaches the submucous areolar tissue. In like manner, the gradually narrowing aperture by which the ulcer eats its way through the sub-adjacent muscular coat, dwindles, as it reaches the peritoneum, to what is little more than a point, corresponding to the centre of the conical ulcer. And it is in this point that the perforation which forms the last event of simple gastric ulceration occurs; generally as the immediate result of the pale yellow slough into which the peritoneum has previously been converted, becoming ruptured or detached, and thus allowing the contents of the stomach to escape into the abdominal cavity.

It is obvious that a progressive increase in the depth of an ulcer would always end in the perforation of the stomach. But this event is in most instances prevented or deferred by the occurrence of adhesion, which, by uniting this organ to some neighbouring surface, obliterates the peritoneal cavity at the base of the ulcer. The peritoneum covering the floor of the lesion undergoes inflammation: its smooth epithelial surface acquiring a dull, roughened aspect; and then becoming the seat of an exsudation of coagulable lymph, which soon fixes and unites it to the adjacent serous surface of any viscus with which it may be in contact.

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[1] These appearances, however, often seem partially due to a *post-mortem* digestion of the ulcerous tissues by gastric juice. (Compare p. 161.)

The frequency of intimate adhesion of course corresponds with that of protraction of the disease; of which protraction this adhesive inflammation may be regarded as equally cause and effect. My own observations enable me to confirm those of Jaksch, who found 22 such adhesions in 57 cases of ulcer; a proportion of about 40 per cent. The site of these adhesions, and the viscus to which they attached and fixed the organ, exhibited a tolerable correspondence with those parts of the stomach already specified as the most frequent situations of the ulcer. Thus of these 22 adhesions, 15 united the pancreas to the posterior surface or lesser curvature of the organ; 5 attached the pylorus or lesser curvature to the adjacent liver; one involved the mesentery; and one the spleen.

But the formation of these adhesions is seriously affected by another cause:—namely, by the movement of the stomach upon the surfaces opposed to it. It is only thus we can explain the rarity of adhesion of the anterior wall of the stomach to the parietes of the belly; coupled (as already noticed) with a by no means infrequent position of the ulcer on this wall, and attended (as we shall see) by an extreme liability to perforation when so placed. And it is interesting to notice, that the situation of the ulcer seems not only to regulate the occurrence of adhesion, but also to affect its structure, and thus to influence its efficiency as a means of protection against perforation. The adhesions which occupy the omentum are often little more than a thickening of the delicate fibrous tissue of the peritoneum by an interstitial deposit of inflammatory lymph; and are of so little avail in warding off perforation, as to be ruptured by very slight exertions or shocks, such as coughing or sneezing with a moderately distended stomach. The adhesions of the anterior surface are often mere threads of imperfectly organized lymph, drawn out into this form from the pasty (compare p. 143) exsudation, by the continual movements of the stomach, so as to constitute a coarse network of fibres, with interstices of various size. While the massy and continuous adhesions which unite the stomach with the liver or pancreas often possess a densely fibrous character, that almost precludes all danger of perforation.

The *duration* of the lesion is very variable, and is, in most instances, rather to be deduced from the symptoms observed during life, than from the appearances found after death. The latter, however, generally permit a conjecture. Thus, when we find a large, shallow ulceration, of irregular shape, unattended by any marks of adhesion on its peritoneal aspect, or by any elevation or thickening of its mucous edge, there is fair ground for presuming it of recent formation. While, conversely, adhesion and thickening around an ulcer, or an exactly circular shape, tend to show that a certain time has elapsed since the first occurrence of the destructive process. The clue sometimes afforded by the symptoms can hardly be alluded to here, save to point out that there is great danger of assigning to an ulcer far too long a duration, in consequence of the liability of the malady to a return. In fact, nothing short of a tolerably complete continuity of the symptoms during a series of years, entitles us to regard an ulcer as really open during the whole of the time. In like manner, unless the symptoms of ulceration during life were very sudden, marked, and persistent, we should hardly be justified in denying that the lesion before us might not have existed before the feelings of illness which first called for medical advice.

Even with these limitations, however, the range of duration is remarkable. In what are certainly exceptional cases, the lesion has been known to be fatal in a few (for example, in ten) days: generally by perforation; sometimes by exhaustion, caused or hastened by vomiting; very rarely by hæmorrhage. But, in the majority of instances, a period of several weeks or months precedes the fatal event. And an extension of this period to years is by no means uncommon. Thus among cases of this kind—possibly frequent relapses, but probably continuous open ulcerations—I find in my notes one of thirty-five years, two of thirty years, three or four of twenty, four or five of fifteen, and several of ten, seven, five, and four years' duration.

The *healing* of such ulcers by a process of cicatrization appears to be far more frequent than is generally supposed. The examinations of Dittrich, Jaksch, Willigk, and Dahlerup, reveal a total of 147 scars, and 156 ulcers, making the proportion of the former nearly equal to that of the latter.

Against such results, it can hardly be alleged that the supposed scars have really been mere local enlargements or thickenings of the mucous membrane, or fibrous deposits in its submucous areolar tissue. While in their favour we may point out, how easily cicatrices of small size might escape discovery, in less careful scrutinies of the mucous membrane of the stomach than those made by these excellent observers.

The cicatrix by which the ulcer heals is therefore, on the whole, about as frequently met with as the ulcer itself. In other words, half the instances of this disease undergo what is probably a spontaneous cure.

The precise details of the process of cicatrization differ with the amount of destruction that has preceded it. Where the lesion has been of small size, and the ulcerative process has not extended deeper than the mucous membrane, the scar is sometimes little more than a mere condensation and thickening of the submucous areolar tissue; and in outline (like the typhoid cicatrix) closely resembles the ulcer that preceded it. But in the majority of instances, it has a more characteristic shape. The gradual contraction of the lymph deposited at the base of the ulcer converts it into a hard, thick, central mass; which gives off cord-like processes, that seem to radiate into the surrounding healthy tissues. The latter are themselves thrown into folds, as the result of the tension which this contraction produces. Where the previous loss of substance has been considerable, this process often seriously affects the shape and capacity of the stomach. In such instances, the cicatrix corresponds to a constriction of the organ, which gives it more or less of an hour-glass shape. And in extreme cases, the contraction amounts to an absolute stricture, which impedes the transit of food, and thus gradually causes great hypertrophy and dilatation of the over-distended segments of the stomach behind the obstruction. Such examples are, however, rare.[1]

The cicatrices which thus modify the calibre and shape of the stomach are generally those of large ulcers, that have remained open for a long period before undergoing the healing process. Hence they are often found associated with adhesion of the stomach to some of the neighbouring organs:—a circumstance which itself aggravates the perils of the gastric constriction, by still further embarrassing the muscular contractions of the stomach, and aiding its changes of shape. In most of these cases, the substance of the adhesion is so inseparably united with that of the cicatrix itself, that it is impossible to distinguish one from the other. Both are indeed composed of the same substance:—a fibrous structure, the elements of which gradually approach, but never attain, the development of the ordinary white fibrous tissue.

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[1] From the cases I have collected, I should conjecture them to form about one in 200 instances of ulcer; or 1 per cent. of the cicatrices by which they are conditionated.

In some cases, the ulcer gives rise to a peculiar dilatation and thickening of the pyloric end of the stomach, so as to convert this part into a kind of pouch, distinguishable through the anterior wall of the belly during life. The details of these cases are insusceptible of a common description, far less of a single explanation; but it seems probable that the accumulation of the gastric contents, which forms the immediate cause of the dilatation and thickening of the coats of the stomach, is chiefly due to a local failure of muscular contraction, itself the result of that destruction of tissue which the ravages of the ulcer bring about. And although a full consideration of the origin of these pouches would lead us too far from our present subject, I may point out that the dilatations or "ampliations" of the stomach caused by gastric ulcer seem to include representatives of two different kinds, each of which may be traced in other gastric maladies. Thus the stricture brought about by the contraction of the cicatrix of an ulcer usually gives rise to an obstruction, such as produces a state of hypertrophy and dilatation, strictly analogous to that variable admixture of these conditions, which often complicates a scirrhus of the pylorus. But in rarer instances, with no material stricture or obstruction, we meet with a more localized dilatation and hypertrophy; engaging, it may be, no part of the stomach save its pyloric sac; and exactly corresponding, by its intestinal extremity, to some unusual destruction of the gastric parietes:—to complete division of its muscular coat; or to a tunnelling of its submucous areolar tissue by the ulcerative process, which raises a bridle or bridge of mucous membrane from off this areolar tissue, and thus constructs a new and abnormal channel for the food, such as allows of the complete disuse and collapse of the natural outlet of the organ. It is to these cases (some of which offer a curious analogy to the ravages of what is usually termed a dissecting aneurism) that I should specially apply the above conjecture; explaining the dilatation and hypertrophy with which they are associated as due to an embarrassment or failure of peristalsis, rather than to any definite occlusion, such as seems to be definitely contradicted by the patulous condition of the ulcerous segment. And by so much as any of them offer a true destruction of muscular substance, they may be regarded as analogous (at least in the locality of this destruction) to that interesting class of dilatations of the stomach in which, with little or no hypertrophy, the tube appears to evince an extreme muscular atony, itself the cause of the dilatation, and often the result of some evidently nervous disease. In other words, not only are hypertrophy and dilatation producible by destruction of the parietes of the tube, as well as by obstruction of its cavity; but the mere paralysis of any segment of the alimentary canal involves its own dilatation, as well as the hypertrophy of the nearest healthy part behind it which can share in that dilatation. In short, as a matter of morbid anatomy, a patulous state of the canal does not disprove the existence of a mechanical obstacle: since such an obstacle is at once created by a mere failure of contraction. All interruption to peristalsis is *pro tanto* obstruction; and if not compensated by hypertrophy, ends in dilatation, as the result of the accumulation and distension which the gradually increasing contents of the stomach or intestine inevitably produce above the obstructed parts. (Compare the remarks on dilatation in Lecture V.)

There are other complications of adhesion and cicatrization which we may dismiss with a very brief notice. In some instances the surface of a broad ulcer becomes completely skinned over, while its firm and extensive adhesion to the neighbouring wall of the belly seems to prevent the complete contraction of the cicatrix. Here (just as in adherent wounds of the stomach, attended with much destruction of its walls) the mucous membrane around the margin of the depression or pit formed by the cicatrix becomes prolapsed and protruded into it, and is thus maintained in perpetual contact with the smooth base of the ulcer.[1] Where the adhesion is smaller and less substantial, it is sometimes drawn[2] out by the constant traction the stomach exercises, so as to form a short and funnel-shaped tube, which is lined by the smooth quasi-serous surface of the cicatrix.

Perforation.—We have already alluded to the simplest and most frequent variety of perforation as being a mere extension of the ulcerative process to the peritoneum, followed by the sloughing or rupture of this delicate membrane, and by the effusion of the contents of the stomach into the peritoneal cavity, with the result of fatal peritonitis. But before passing on to consider those modifications of this process which have sufficient pathological interest to deserve a brief notice, we may point out a few general considerations respecting the event to which the term "perforation" is generally applied.[3]

The history of a large number of such cases has been nearly as follows. A person, often a young and apparently healthy female, in other instances dyspeptic or chlorotic during a variable time, is suddenly attacked, soon after a meal, with excruciating pain in the belly, followed by all the symptoms of peritonitis, speedily ending in death. Such a rapid transition from apparent health to agonizing pain and death naturally excites much attention; and sometimes leads to the suspicion of poison.

But though the interest thus attracted to these cases had given rise to much speculation respecting their nature and origin, still it had not led to their being collected and sifted in such a manner as to admit of valid conclusions respecting many of their details, until, four years ago, I brought together from various sources 234 instances of such perforation. The information derived from these cases suggested the following conclusions.

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[1] It is not impossible that the friction of such an abnormal surface may favour that recurrence of the ulcerative process which appears often to obtain in such cases.

[2] A process of elongation which is also seen in peritoneal adhesions of other abdominal organs.

[3] To avoid misunderstanding, I use the term "perforation" in its ordinary sense:—namely, that of such a penetration of the walls of the stomach as allows the effusion of its contents into the peritoneal cavity. Of course, pathological accuracy would require us to regard perforation as having taken place in all cases in which these walls had been penetrated, no matter what viscus or exsudation might stave off the ordinary consequences of this solution of continuity.

As regards the *frequency* of perforation in the course of gastric ulcer, its occurrence is so far exceptional, that not more than 1 in every 7 or 8 cases of this lesion (1 in 7·45 = 13·4 per cent.) terminates in this way.

The *sex* of these cases of perforation offers nearly the same proportion as that which we have deduced for the ulcer generally. The 234 instances collected consist of 160 females and 74 males;—a ratio of about 2 to 1.

In respect of *age*, however, there is a remarkable contrast between the perforating ulcer and the ulcer generally. The latter we have found especially to affect the periods of middle and advancing life, with a frequency that gradually increases up to the extreme age allotted to Man. But the perforating ulcer not only selects another period of life, but exhibits a marked contrast of age in the different sexes; the period of life in which it is most liable to occur being quite a different epoch in the male and in the female.

Thus, from a careful analysis[1] of these cases and their ages, we deduce the following conclusions:—

The total risk of perforation during the whole of life is nearly equal in the two sexes.

The general or average liability to perforation, in both sexes collectively, undergoes a constant decrease as life advances; even though the liability to the ulcer itself is just as constantly on the increase. The amount of these two changes of risk is such that we may estimate the risk of ulcerous perforation of the stomach as gradually sinking to one half its former amount, as the individual lives on from the age of thirty to that of seventy; while the risk of being attacked by gastric ulcer is gradually rising to double.

The distribution of this liability to perforation over the whole of life varies materially in the two sexes.

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[1] The details of this analysis will be found in the "British and Foreign Medico-Chirurgical Review" for January, 1856; and in the monograph already cited.

In the female about half of it (or the whole excess of cases over those seen in the male) fall upon the years between the age of 14 and 30: and one-third upon the six years between 14 and 20. In the male it is a constant proportion up to the age of 50, and diminishes but little up to that of 70. Hence the average age of the subject of perforation also differs in the two sexes, being 27 in the female, 42 in the male.

Such a remarkable liability to perforation at this epoch of female life, naturally suggests the physiological events of this period as more or less immediately causing the occurrence. But not only do the proportions of older females, and of males, who succumb to this accident, afford abundant evidence that it is essentially independent of any such cause, but the circumstances of many of these cases themselves inculcate great caution in coming to any definite conclusion of this kind. Some of them are expressly mentioned as not having arrived at puberty: others are recorded to have menstruated regularly, and even profusely: and finally, one of the most characteristic instances occurred in a person who, though supposed to be a female, was proved by a careful necropsy to be devoid of ovaries; and therefore, physiologically speaking, alike incapable of menstruation, or of any conceivable disorder of this function.

And whatever the relations which the various symptoms of such cases bear to each other; or however expedient it may be to regard these, with their age and sex, and their liability to perforation, as constituting them a special group; we know nothing at present which would justify us in regarding them as pathologically distinct from others. On the contrary, there is every reason to affirm, that in a large number of the females who become the subject of the lesion at this epoch of life, it has precisely the same origin, course, termination, and appearances as it would have in any other person, whether old or young, male or female.

The maximum and minimum ages scarcely deserve notice. The oldest case I am acquainted with is that of a man of eighty-two; the youngest a girl of eight, and a boy of nine years.

As respects the *situation* of the perforating ulcer, a careful contrast of large numbers of this and of the non-perforating lesion indicates that this circumstance has a remarkable influence on the accident of perforation. For though the posterior surface of the stomach is the part most frequently the seat of ulcer, it is one of those least liable to perforation: while conversely, the anterior surface, though much more rarely occupied by the ulcer, is yet one of the most frequent sites of perforation. Indeed, if we may rely on the cases thus analysed, it would appear that the proportion of perforations to ulcers is such, that of every 100 ulcers in each of the following situations, the numbers which perforate are:—on the posterior surface, about 2; the pyloric sac, 10; the middle of the organ, 13; the lesser curvature, 18; the anterior and posterior surface at once, 28; the cardiac extremity, 40; and the anterior surface, 85. In other words, in the first of these situations the probabilities are about 60 to 1 against perforation; in the latter they are 6 to 1 in its favour:—a fifty-fold increase of risk. The relation of these surfaces to the occurrence of adhesion, as well as to the nature of the tissue which effects it, has already been pointed out (p. 139).

We may perhaps notice the anomaly implied in the above statement—that while the ulcer of the anterior surface perforates about 85 times in 100 cases, the double ulcer of the anterior and posterior surfaces only does so about 28 times in 100, or less than one-third of that proportion. Now we can scarcely suppose that the usual tendency of the anterior ulcer to undergo perforation is directly diminished to such a great extent by the presence of another ulcer on the posterior surface. We are therefore left to conjecture some original or specific difference in the ulcerative process by which this anterior ulcer is produced. The curiously exact apposition of the two ulcerated surfaces in many of these cases strengthens this suspicion: and suggests that, in some of these instances, the anterior ulcer is preceded and caused by the posterior one, the perpetual contact of which with the opposite gastric surface gives rise to its secondary ulceration, much as a similar contact with the original ulcer often leads to ulceration or suppuration of the liver, pancreas, and spleen. That such a secondary ulceration should be less active, less intense, and therefore less likely to perforate, would not be very surprising.

The age at which perforation occurs seems quite uninfluenced by the site of the ulcer: the average age of 67 cases of perforating ulcer of the anterior surface of the stomach being exactly that of perforating ulcer in general, in each of the two sexes (see p. 145).

As respects any influence exercised by sex on the situation of the perforating ulcer, it would seem that, on the anterior surface of the stomach, this lesion is twice as common, and on the cardiac end five times as common, in the female as in the male; in whom, conversely, the pyloric extremity appears to be perforated three or four times as often as in the female.

There can be no doubt that a mechanical tension of some kind is generally the immediate cause of perforation, by rupturing the thin film of tissue to which the ulcer has already reduced the coats of the stomach. Indeed, in a great majority of instances, the occurrence will be found to have taken place immediately after a meal; or in other words, with a distended stomach. And in less frequent cases we may find evidence of other mechanical agencies. Thus, perforation has been brought about by vomiting; by the abdominal pressure on the stomach in the act of defæcation; by the rupture of a delicate adhesion of the omentum to the anterior wall of the stomach, in the act of sneezing; by the sudden constriction of the waist by a tight belt; by the jolting of a cart; and by the displacement of a kind of plug formed by the adherent omentum in contact with the exterior of the ulcer.

Complete perforation of the walls of the stomach is generally accompanied by the sudden effusion of more or less of its contents into the cavity of the belly. But the degree and extent of this effusion are liable to great variety. One or two instances are recorded, in which the accident has given rise to none of its ordinary symptoms, and has been followed by no appearances of peritonitis in the dead body. In some of these cases it seems very doubtful whether the perforation was really complete during life; or whether the aperture observed may not have been caused by the solvent action of the gastric juice after death upon the film of peritoneum forming the base of the ulcer. In others, the anomaly appears to have been due to the state of the patient having prevented the access of all symptoms; the perforation having occurred during the approach of death from the exhaustion produced by the ulcer, or by some independent disease. In equally rare instances the stomach appears to have been retained in such close and immovable apposition to the wall of the belly by the abdominal pressure (see p. 51), that scarcely any of its contents have escaped, save a small quantity of a clear fluid, which has (as it were) filtered between the surfaces of contact, and lit up the fatal peritonitis.

In other instances, the effusion of the gastric contents is confined to the immediate neighbourhood of the perforated spot; and the inflammation which they excite, being equally limited, may be distinguished as circumscribed peritonitis. These cases are of course less immediately fatal than those in which a wider extent of the serous surface is involved in the inflammation. But the patient often survives the first shock of the accident, only to succumb to the combined effects of peritonitis and gastric exhaustion. In other instances, a different result obtains: the portion of the peritoneal cavity circumscribed by the inflammation continues to suppurate, and is thus gradually converted into a chronic abscess, which finally discharges its contents at some point or other of its exterior. There are about twenty cases of this kind on record. Their age and sex give me precisely those averages which we have already deduced for the accident of perforation generally. Their other features are almost as easily summed up.

As implied above, the circumscribed character of the inflammation appears due to the limited diffusion of the gastric contents; which, so far as they reach, seem always to excite this process. What restrains them in such narrow bounds it is not always easy to specify. Sometimes, however, it is evidently a deposit of lymph, caused by extensive adhesive inflammation around the ulcer prior to its perforation. Sometimes the delicate omentum forms a septum which bounds the lower surface of the sac. Sometimes the transverse colon lends a more or less temporary aid to the process: or a casual coil of some other part of the intestinal canal affords a similar assistance. Sometimes the aperture in the peritoneum seems too narrow to allow of more than an inconsiderable leakage, such as spreads very slowly on all sides of it. In any case, the apposition already mentioned as effected by the abdominal pressure is soon aided, first by the distension and immobility[1] which peritonitis brings about, and next by a rapid effusion of lymph:—two circumstances which combine to render such localizations permanent, and to seal up the mischief within the limits to which the above mechanical causes tend for the time to confine it.

The opening of a gastric ulcer into the chest is generally accomplished by the mediation of such an abscess: less frequently by a recurrence or extension of the ulcerative process destroying an adhesion between the stomach and the diaphragm. The penetration of this septum has been known to be followed by instantaneous suffocation. In most instances, however, the fatal event is preceded by an interval, during which gangrene of the lung or other pulmonary lesions arise. The pericardium is very rarely opened.

The communication of the ulcerous stomach with the exterior of the belly by a fistulous aperture seems, in most of the instances[2] recorded, to have been the result of a similar abscess, which has pointed and burst like an abscess of the liver.[3] The gastric fistula, once established, either kills by exhaustion, or (what seems more usual) gradually closes, just like the artificial fistula established in animals for the purpose of physiological experiment. Subsequently to its closure, the adhesion of the stomach to the anterior wall of the belly is sometimes drawn out into a fibrous cord, which is occasionally excavated by a funnel-shaped cavity, that is itself continuous with the inner surface of the stomach at its broad base, and is lined by a smooth membrane of a serous aspect.

The communication of the stomach with other parts of the alimentary canal, as the result of gastric ulcer, is generally

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[1] Compare p. 66.

[2] About six or eight undoubted cases of this kind are all I can recollect to have met with.

[3] In one case of this kind, fatal by hæmatemesis, the abscess in front of the stomach communicated with a suppurating cavity which occupied the areolar tissue of the rectus abdominis muscle.

independent of any such abscesses. The ulcerated part of the stomach becomes attached by lymph to some portion of intestine in contact with it: and a mere extension of the ulcerative process successively removes the coats of the stomach, the lymph, and the coats of the intestine, where these are united to each other. As regards the situation of such abnormal apertures, there are one or two cases recorded in which the stomach has opened into the neighbouring segment of the duodenum; and about ten in which a similar communication has been brought about between the stomach and colon. The comparative frequency with which this segment of the canal is selected as the site of the communication, is of course referable chiefly to its situation and size.

The frequency with which ulceration implicates the liver and pancreas cannot be exactly estimated. But in the large proportion of ulcers (70 per cent.) occupying the posterior surface and lesser curvature of the organ, these viscera are often attacked. The cardiac extremity of the stomach is very rarely the seat of the ulcer; and hence penetration of the spleen belongs to the rarer sequelæ of the malady. And as none of these viscera can be excavated by the ulcer unless they have been previously attached to the stomach by adhesive inflammation, the perforation which their excavation really implies, has a much less dangerous character than where it opens the peritoneal sac. The chief danger, indeed, seems to be that of hæmorrhage; either from the arteries which course along the upper border of the pancreas, or from the smaller vessels which ramify in the substance of the liver and spleen for their supply. Gangrene of the two latter viscera is, however, by no means infrequent. And of course, the adhesions alluded to may themselves at any time become the seat of further ulceration; which, without causing any new perforation of the coats of the stomach, may extend through the more or less organized lymph into the cavity of the belly and thus cause a fatal peritonitis.

Hæmorrhage is another incident of ulcer of the stomach which deserves a special mention. We have already noticed that discharge of blood from the mouth or anus, which generally follows a considerable gastric hæmorrhage, as constituting one of the most frequent and important symptoms of this lesion. So that we have now only to describe its mode of production, and its bearing on the termination of the malady, as a cause of death.

In respect to the sources of such hæmorrhage, we may distinguish four: which generally come into operation at different dates of the malady; and which certainly have a very different influence on its course.

In the first place, analogy and observation coincide to indicate, that the congestion which often attends the commencement of ulceration of the stomach may give rise to a hæmorrhage from the vessels of the mucous membrane. But since, without any existing breach of surface, we cannot define the case as ulcer, while, with it, we can rarely exclude the possibility of this lesion having been the source of the bleeding, such a cause of hæmorrhage is rather to be admitted as a probability, than stated as a fact.

The progress of the ulceration itself determines the three following varieties of hæmorrhage. As the breach of surface gradually involves the vascular mucous membrane, it successively erodes a vast number of vessels:—at first mere capillaries; then the minute arteries and veins from which these capillaries ramify; and lastly, the small vessels of the arterial and venous plexuses which occupy the sub-mucous areolar tissue. The hæmorrhage permitted by these numerous solutions of continuity is probably often arrested at once by a coagulation of the blood within the open extremities of the eroded vessels. More frequently, however, it gives rise to a slow drain of blood in very moderate quantity. This, as it flows, mingles with the secretions and contents of the stomach, and gradually undergoes the usual changes of blood when exposed to the digestive action of the alimentary canal: exchanging its crimson colour for one which is almost black, and exhibiting (if in sufficient quantity for such a change to be visible) a viscid or tarry consistence. In rare instances, the quantity of blood thus set free is much more considerable, and closely imitates the more important hæmorrhage which forms the third variety. In such cases we may conjecture the hæmorrhage to be increased by a sudden congestion of the ulcerous stomach.

The third and most serious class of hæmorrhages is one in which the bleeding comes from a large artery of the stomach. Consistently with such a source, it will generally be found to occur at that later stage of the gastric ulceration, when, after penetrating the mucous and the muscular coat, it reaches that interval between the latter and the peritoneal coat in which these vessels run; or when, in the case of an ulcer of the posterior surface, it eats into the adhesion, fixing it to the pancreas, so as to erode the splenic artery which courses along the upper border of this gland. (Compare Fig. 8, p. 18.)

The blood poured out by such hæmorrhage often retains the characteristic marks of its arterial source after its expulsion by vomiting. In other instances, it assumes a dark and clotted appearance; in a degree varying with its amount, the rate of its flow, the gastric contents with which it has been mixed, and similar circumstances. In some cases it is rapidly effused in such vast quantity that death ensues almost instantaneously; and it is only at the necropsy that its cause is revealed, in the shape of an enormous mass of clotted blood, distending the stomach and a variable extent of the intestinal canal.

Such hæmorrhages have one feature in common with perforation—namely, that they generally occur soon after a full meal. The mechanical influence of gastric distension in disturbing the eroded vessel is too obvious to require any comment. It seems to be assisted by that afflux of blood to the stomach which attends its digestive act. This view is confirmed by several cases, in which the hæmorrhage has followed a violent mental emotion. Doubtless, also, the occurrence of both hæmorrhage and perforation soon after meals is greatly aided by the solvent effect of the gastric juice then poured out.

As regards the frequency of these larger bleedings, they are often absent during the whole progress of the ulcer. In many cases, again, they do not cause death. And even when they are fatal, it is rarely by a single attack.

In many instances the necropsy has shown a peculiar condition of the vessel, such as quite explains the intermittent, though repeated character of these hæmorrhages. The ulcer has cicatrized over its whole extent excepting that point in its centre which is occupied by the eroded artery. And the calibre of this tube has been found filled by a clot, the casual detachment of which from time to time has evidently been the cause of the hæmorrhage.

The fourth kind of hæmorrhage has already been alluded to, as forming what is strictly a sequela of perforation, and as consisting in the erosion of vessels occupying the substance of the liver, pancreas, or spleen. These vessels are generally the small arteries and veins of the proper substance or parenchyma of the above glands. And in the moderate hæmorrhage to which their breach of continuity usually gives rise, the blood effused undergoes a change similar to that noticed in the second variety.

As regards death by hæmorrhage in ulcer of the stomach, its frequency appears to correspond with a proportion of 1 in 20—30, or 3—5 per cent. It appears to be nearly four times as frequent in the male as in the female. The average age of the patients is the same in both sexes:—about forty-three years. Unlike the perforating ulcer, the situation of the ulcer causing such hæmorrhage seems no way peculiar. The protracted duration and great depth of these lesions are their chief features; the influence of which in causing such hæmorrhage is obvious enough. The source of the hæmorrhage is almost invariably one of the three large arteries of the stomach, or some primary branch of them. Which of the three, is dictated chiefly by the situation of the ulcer; the posterior surface being the usual seat of ulcers eroding the splenic artery; while, for equally obvious reasons, it is in or near the smaller curvature that we generally find the source of an erosion of the coronary or superior pyloric vessel. The splenic is the artery which suffers most frequently; its proportions (about 55 per cent.) being referable, not merely to its larger size, but to its closer fixation, and to the greater frequency of the ulcer on the corresponding surface of the stomach. The rare instances of fatal hæmorrhage from the eroded substance of the liver or spleen, show a similar contiguity of the lesion. (Compare pp. 18, 20, Fig. 8.)

Concerning *exhaustion* or starvation, as one of the terminations of gastric ulcer, I can only tell you that there is no doubt, both of its frequent occurrence, and of its being often amenable to treatment. You will easily understand why I cannot offer any precise numerical estimate of its frequency, such as I have adduced for the other modes of death in this malady. Considering, for instance, that it is often not so much difficult as impossible to analyse this state in any given case—to separate the direct influence of the ulcer on the digestive powers of the stomach from the results of the vomiting it excites, or from the debility produced by moderate hæmorrhage—you will not wonder if I offer you conjectures rather than large and accurate facts like those I have elsewhere set before you.

All the information I have been able to collect, suggests or confirms the following propositions. Even in the middle-aged subjects of this lesion, death by exhaustion claims somewhat about three, four, or five per cent. as being evidently the chief (if not the sole) cause of death. Indirectly, it is far more frequently the cause of this event: by rendering unusually fatal some one or other of those illnesses which, in the course of years, few persons altogether escape; and in which any serious impairment of one of the most important structures and functions of the body must suffice to turn the wavering balance of life or death.

And as regards the older subjects of gastric ulcer, I would assert a very similar proposition. Remember, I would say (not as a moralist, but as a physician), that we all bear with us the seeds of a mortal disease, to which, even if we escape all other maladies, we must inevitably succumb—old age. Whether for prognosis or for treatment, the influence of age on the course and event of disease cannot be too sedulously borne in mind. Certainly in this malady it is all-important. Despite the small absolute number of these lesions found at advanced ages—a fact which doubtless renders their comparison with the small number of such persons living, and the large proportion it affords, somewhat less trustworthy—it is impossible to doubt the substantial accuracy of the conclusion to this effect previously deduced (p. 133). Both from the foregoing wide inquiry, and from careful clinical observations extending over many years, I cannot but conclude that mere age, as such, has a remarkable influence, not only in producing this disease, but also in aggravating its effects when produced. In about thirty per cent. of all cases of gastric ulcer, it is probable that the lesion gives rise to a process of marasmus or partial exhaustion, which anticipates, hastens, or increases that gradual failure of the nutritive functions which is one of the most essential elements of death by old age.

The relation of gastric ulcer to cancer of the stomach we shall consider in treating of the latter disease. At present it need only be said, that though the former is doubtless capable of provoking a cancerous deposit, yet the estimate of some good observers—that of every hundred gastric ulcers, ten exhibit concurrent cancer of other organs—must be partly explained by the great difficulty sometimes found in distinguishing two lesions which, whatever their pathological relations, can generally be distinguished on anatomical grounds.

The ordinary combinations of ulcer with cancer of the stomach seem to be chiefly limited to a cancerous degeneration or deposit that involves the hard brawny mass which we have already (p. 135) noticed as generally present, in variable quantity, in the base or periphery of an ulcer of long standing. In rare instances, in which the whole of the substance around the ulcer is converted into a cancerous excrescence, it is chiefly by the shape and other characters of the ulcerated depression that (in the absence of any history of the case during life) we should discriminate between the cancerous degeneration of the hard margin of an ulcer, and the ulceration of a growth originally cancerous. But in a majority of such cases the decision is less difficult. Indeed, a not infrequent form of such a combination appears to be that in which a fungus (generally a bleeding one) shoots up from the basis of a gastric ulcer, the characters of which are in all other respects those usually seen in this lesion.

Lastly, as regards the complication of the gastric ulcer with lesions of other organs, it appears from the statements of Jaksch, Dittrich, and Engel, which refer to some hundreds of ulcers, that pulmonary tubercle is present in about nineteen or twenty per cent. Jaksch and Engel also concur in estimating the frequency of pneumonia and pleurisy at about twenty-seven per cent. Engel finds ten per cent. to be preceded by syphilis. While both from the records I have examined, and from my own personal experience, I deduce the general conclusion; that the lesion is associated with a large number of diseases, in proportions much more akin to those of their known frequency than to any other of their circumstances.

Such a conclusion is of course quite compatible with the suggestion, that the long and exhausting malady which gastric ulcer produces, and which is the expression of a serious lesion in so important an organ as the stomach, perhaps predisposes the constitution to a variety of other diseases. It is chiefly as regards the special maladies that the contrast above hinted at becomes indispensably necessary. For example, the large percentage of phthisis renders it one of the most frequent complications of gastric ulcer, according to all observations hitherto on record. But the significance of such a proportion must evidently depend, not so much on its absolute amount, as upon a comparison of this with the average share taken by phthisis in the mortality from all causes. In other words, if the gastric ulcer really had any direct influence as a predisposing cause of phthisis, we should expect to find, not merely a large absolute number of ulcerous cases dying with this malady, but such a proportion as would considerably exceed the average ratio of the deaths by phthisis to those from all causes indifferently. But since the deaths by phthisis, in persons of both sexes above the age of twenty, amount to rather more than eighteen per cent. of the deaths from all diseases, the statement that twenty per cent. of the cases of gastric ulcer die of phthisis, is one which, even if confirmed by a wider series of observations, will by no means justify us in assuming any such causation.

The *ætiology* of the gastric ulcer has hitherto been a subject of conjecture rather than for inductive inquiry. And while the facts I have adduced with such an object correct some errors and suggest various interesting conclusions, still they are far from affording a complete explanation of the causes of this lesion.

(1) There can be no doubt as to the physiological circumstances which predispose to this disease. Old age, privation, fatigue, mental anxiety, and intemperance so frequently coincide with it, that we are fully entitled to regard them as its (more

or less immediate) causes in a large proportion of cases. Of these causes, advancing age seems to be that which is most distinct and indisputable, and which rests on the broadest numerical basis of facts. But the careful clinical study of the malady leaves just as little doubt with respect to poverty and intemperance.

(2) We have found reason to qualify the ordinary notion with respect to the influence of the access of female puberty, and its attendant disturbances of health, in the production of the gastric ulcer. At least we have seen that this epoch predisposes, not so much to the occurrence of this lesion, as to a peculiar character and termination of it; that it is a want of reaction, and a tendency to perforation, rather than a proneness to ulceration, which our existing data would entitle us to assert. And even should this opinion be hereafter modified by a larger series of cases than those which have led me to this conclusion, there will still be such a preponderance in the total formed by the gastric ulcers of the male, and of the middle-aged and aged female, as to exclude the above group from any general significance in the ætiology of the disease.

(3) Respecting the influence of the puerperal state as, well as of lactation, I can only say that, in the instances in which these conditions have been coincident with gastric ulcer, the connexion has seemed to me explicable by well-known physiological laws. In the former state, for instance, the ulceration has appeared to be a relapse or recurrence of a similar morbid process suspended during pregnancy: in the latter, to be partially referable to the exhaustive effect of suckling on a feeble constitution—perhaps sometimes aiding an analogous relapse. And I know of no facts which would justify the supposition of any more direct influence being exercised by either of these conditions in the production of gastric ulcer.

(4) The specific diseases with which the gastric ulcer has been observed to concur, seem far less influential than the above physiological conditions. Pulmonary tubercle, pleurisy, pneumonia, syphilis, ague, and fever, are the maladies most frequently revealed by the necropsy and the previous history of cases of gastric ulcer. But it has already been stated

that the percentage of tubercle does not seem to exceed its average in all persons indifferently. And the same statement will probably apply to some of the other diseases above mentioned. It would seem, too, from my own inquiries, that these cases of gastric ulcer do not exhibit that family history of phthisis which we should expect, on the supposition of there being any causal connexion between the two diseases, and which we certainly find in pulmonary tubercle.

(5) Many of the peculiarities of the gastric ulcer appear to be explicable by the circumstances of stomach-digestion. Ulceration having once taken place, the ulcer is prevented from healing, and even increased, by those great and sudden alterations in size which the organ undergoes at different periods of digestion; by the chemical and mechanical irritation of the food; and by the solvent action of the gastric juice upon the languid tissues which form the periphery and base of the ulcer, or upon the scarcely organized lymph which has been poured out in this situation.[1]

But in respect to the ætiology of the lesion, such explanations are useless. For, in explaining what happens after ulceration has once taken place, they afford no answer to the inquiry why ulceration takes place at all; and especially why it singles out this particular organ.

(6) It may be said, however, that the continuance and extension of the gastric ulcer form its chief characteristics: the cicatrices of ulcers are frequently found in the intestine; and that the peculiarities of the ulcer of the stomach, as contrasted with the ulcer of any other part of the alimentary canal, consist in its long duration, its gradual enlargement, and its reluctance to cicatrize.

It may seem hard to throw discredit upon an explanation possessing so much practical usefulness as this certainly does. But it is the duty of clinical research to subject to strict scrutiny every view not already established on an irrefragable basis. Now, not only have we no series of facts (such as the careful records of systematic necropsies would afford) sufficient to establish the propositions these statements imply, but all the information as yet at our disposal inculcates the greatest caution in receiving them. The ulcer of the stomach appears to cicatrize, probably in many cases without medical treatment directed to this end, in about fifty per cent. of its total numbers. It does not seem to perforate oftener than about once in eight cases. No doubt, the class of intestinal ulcers, as a whole, offers a large percentage of cicatrices, and a smaller of perforations, than the above numbers. But the truth of the above propositions assumes an amount of these numerical differences such as we have at present no right to assert.

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[1] Compare Dr. Budd's "Lectures on Diseases of the Stomach," *Medical Times*, 1853, vol. vi., p. 618.

Further, if we examine into the details of those lesions which would be grouped under the terms “gastric” and “intestinal” ulcers, we shall find reason to attribute still less value to the above numerical differences. For we have seen that the majority of ulcers of the stomach cannot, in the present state of our knowledge, be traced to any specific constitutional disease. While we know that almost all those intestinal ulcers we are now contrasting with them merely form the local expressions of a general malady; by the nature, date, and duration of which they are themselves dictated and regulated. The typhoid fever reaches its term, and the exulcerated agminate follicles generally heal over; the attack of dysentery subsides, and its ravages in the large intestine are more or less repaired; the phthisical cachexia continues and increases, and therefore the ulcers it has produced also persist and extend. It is not impossible that ulcer of the stomach may be sometimes due to a chronic vice in the organism which dictates its occurrence, duration, and cessation, like the typhoid, dysenteric, or tubercular states of the constitution respectively. But in default of all evidence to show that this represents a frequent form of the lesion, there is, at any rate, sufficient difference between the two classes of “gastric” and “intestinal” ulcer, to forbid the unqualified reception of any pathological theory which regards them as alike in all save their mechanical and chemical circumstances. Besides, it seems very easy to overrate the influence of the circumstances above alluded to, in retarding the progress of cicatrization in the ulcer of the stomach. Wounds of this viscus heal with great facility, not only in the domestic mammalia usually selected for the purposes of physiological experiment, but in the healthy human subject. Indeed, in both man and animals, fistulæ of the stomach seem to progress naturally towards closure and cicatrization, although placed under conditions apparently less favourable to such a result than many other fistulæ. While, as already mentioned, the ulcer itself heals with a frequency that can scarcely be explained by any theory which makes the circumstances of the digestive process so principal or special a cause of its persistence. Lastly, every organ is adapted to its circumstances, and is generally organized to resist any unusual exposure which these may imply.

(7.) The morbid appearances which inaugurate and attend the ulcerative process would probably throw some light on its causes. But unfortunately these still remain almost unknown to us. Direct observation is hardly possible. Analogy has little value. And the scanty and imperfect details adducible from both of these sources, are scarcely even consistent with each other, much less applicable to any single view of the ætiology of gastric ulcer.

It may, however, be noticed, that necropsies sometimes show ulcers of the stomach associated with more or less circumscribed patches of congestion, ecchymosis, or extravasation, which are regarded as the beginnings of similar lesions in the neighbourhood of these ulcers. A similar (though quite distinct) class of appearances, constituting the "hæmorrhagic erosion" of Rokitansky, is believed by him to represent the mode by which the ulcer commences. But, to say nothing of the comparative rarity of these appearances in conjunction with the gastric ulcer, the irregularities which often affect the distribution of blood after death in the digestive canal (see Lecture II.) suggest great doubt as to how far the first class of phenomena form decisive evidence of commencing ulceration. And the last is not only partially open to the same objection, but appears to be quite a distinct disease from gastric ulcer, capable of continuing an indefinite time without showing the slightest disposition to merge into it, and exhibiting a different train of symptoms. (See p. 101.)

In still more exceptional cases we find other phenomena which suggest similar doubts. A distinctly oval or circular depression has been found in the neighbourhood of an ulcer of the stomach; or the mucous membrane has exhibited an appreciable softening of the same size and shape—once or twice in conjunction with a reddish or darkish tinge of discoloration. Here, again, we have no sufficient proof that these depressions or softenings would have become ulcers, or even that they existed during life. Analogy, however, affords a strong presumption that some such process may originate the gastric lesion; by sometimes exhibiting an appearance of this kind in the duodenal ulceration consequent on severe burns.

The extremely variable appearances which we have found associated with different examples of the lesion, confirm (or even increase) the doubts suggested as to its commencement by these remarks. For practical purposes, they distribute the ulcer into two classes; respectively attended by appearances of reactive inflammation, and devoid of them; but merging into each other by infinite shades of admixture. And even in the best-marked examples of the latter kind—in which, as already (pp. 134, 136) mentioned, there seems to be a mere removal of tissue—we are unable to describe (or even to name) the process which effects it. We may call it "destructive absorption;" but we cannot decide whether, even during life, the destruction of ulceration may not be aided by a solution effected by the gastric juice, or whether the absorption by the surrounding vessels is complete enough to exclude all effusion of the dissolved tissues into the gastric cavity. While as regards the appearances seen by us after death, it is often impossible to decide how much of the soft, flaky mass which forms the floor and margin of such a "simple" ulcer, really occupied this situation before death; and how much is to be attributed to the ordinary processes of gastric decomposition. (Compare pp. 75 and 136.)

It would therefore seem that our existing information not only affords no single theory of gastric ulcer, but definitely negatives any such view. Judging by the really vast array of facts now collected, not only is there every reason to deny the existence of any specific disease that can lay claim to the title of "*the ulcer of the stomach,*" but the numerous varieties in the form, progress, situation, number, and termination of this lesion, seem to find their parallel in the causation of the malady, both as regards the organism generally, and those first departures from the normal state which inaugurate the local mischief. In short, we have no more right to talk of *the* ulcer of the stomach than of *the* ulcer of the leg; no more reason to assume an invariable commencement of the gastric ulcer by hæmorrhage, or by softening, or by a sub-mucous deposit of lymph, than we have to restrict the beginning of what is evidently a similar process of ulceration in the limb to an ecchymosis, a pimple, a superficial abscess, a burn, or a varicose vein. And just as daily experience assures us that external or cutaneous ulcers may begin by either of these lesions—may have, that is, either of them as its immediate and conditionating cause, and yet retain a general identity of that ulcerative process to which they are chiefly due—so not only do these very facts afford us fair grounds for supposing a similar diversity in cases of ulceration affecting the stomach, but all that we have been able to glean respecting such ulceration confirms this analogy. The variety of diseases with which it appears to be connected, the equally numerous and diverse physiological conditions that favour its occurrence, can only thus be explained. Ague, fever, the ingestion of alcoholic irritants, or the vascular disturbances of female puberty, might perhaps be supposed to facilitate or cause ecchymosis. But the conversion of this hæmorrhage into an ulcer implies a process of destruction, which no mere extravasation would explain; and the causes of which are but partially traceable in the cachexia of the aguish or feverous convalescent, the toxæmia of the drunkard, or the want of due light and air to which many of the future mothers of our nation are condemned. While the influence of old age, privation, or fatigue, which throws little or no light on the precise local change that ushers in the ulceration, exactly concurs with the efficacy of these circumstances in the production or promotion of ulceration elsewhere. In like manner, to grant that the circumstances of digestion often retard the healing of a gastric ulcer—and the marked effect of treatment specially directed to these circumstances proves no less—is to concede nothing more than what (*mutatis mutandis*) we may verify for an ulcer of the leg; in which the same event is equally under the influence of such physical circumstances as exercise, posture, pressure, and the like. Nay, more, the analogy not only applies to these details, but appears to illustrate some of the remarkable facts previously elicited. For example, we found reason to conclude, that the epoch which immediately follows the access of puberty in the female imparts to the gastric ulcer peculiar characters, best summed up as a deficiency of that inflammatory reaction that generally engages the base and margins of the lesion. And a similar character has been observed in the ordinary cutaneous ulcer at the same period of life; associated, too, with an analogous cachexia, and with an equal disturbance of the menstrual flux.[1] Indeed, the resemblance of the two lesions is completed by the facts, that in both the ulcer precedes the amenorrhœa, and often exhibits an aggravation of its symptoms at the menstrual periods.

We are left to notice certain considerations which, though quite independent of any such theory of gastric ulcer as those just demurred to, are nevertheless scarcely less true, and far more useful, than would be the most dogmatic view of this morbid process. That ulcerations, sufficiently large and continuous to merit the title of "ulcers," are, on the whole, far more common in the stomach and duodenum than in any equal area of intestinal mucous membrane, is a statement which may be as broadly expressed, as it is absolutely implied in many of the foregoing pathological data. And difficult (or, indeed, impossible) as it has hitherto been to explain how the gastric juice—the only agent assignable to this precise locality—can originate (as it notoriously does maintain and enlarge) gastric ulcers, yet the exact co-extensiveness of ulceration and gastric juice—in other words, the coincidence in place of the lesion and the solvent—has of course suggested a causal relation between them.

To the statement of this coincidence I may add that of another, which repeats, in perhaps a more exact form, the same contrast of comparative frequency. It may be confidently asserted that the cancers of the stomach and duodenum (but especially the former) ulcerate much more frequently, as well as much earlier, than do those of the various other parts of the alimentary canal. This statement must of course be taken in such a strict pathological sense as would allow us to find its illustrations in outgrowths of analogous form, species, and duration, and not in cancers essentially so diverse in many of these respects, as are ordinary cases of cancer of the rectum contrasted with those of the pylorus.

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[1] Critchett "On Ulcers of the Lower Extremities," p. 107, *et seq.* London. 1849.

The abrupt gap thus left in the ætiology of gastric ulceration, by any careful and conscientious survey of its numerous aspects, need, I think, no longer be the main feature in its picture, but may be almost regarded as filled up. It has, perhaps, been long bridged over by any who could complacently assume that the two well known and striking facts above mentioned explained each other, without feeling bound to adduce one tittle of evidence as to *how* they did so.

Hitherto no one has been able to surmise how the gastric juice *begins* any destruction or solution of the stomach: to assign, that is, any reason for such a process of solution occurring with even the frequency we have verified for gastric ulceration, and yet not occurring in every person, not to say every vertebrate animal, to a degree downright incompatible with health and life. This has been the *reductio ad absurdum* to which any explanation based exclusively upon the above coincidence would logically have led. But Dr. Pavy's observations,[1] while they remit to further, and it may be very different researches, the task of deciding how far an undue acidity of gastric juice may inaugurate or enlarge an ulcer, have the broad substantive merit of indicating that any such extreme congestion as can retard and (however temporarily) arrest the flow of blood through a given area of gastric mucous membrane, may thus allow this energetic secretion to cause what, with more or less of adjacent reaction, constitutes an "ulcer of the stomach."

It is neither necessary nor desirable to carry out, in all its natural consequences, this invaluable piece of information. But that it throws light upon many of the details of gastric ulceration collected in the preceding pages, will be obvious, I think, to those who will take the trouble to glance over them with such a clue to their import. Stagnation of gastric blood, whether as the physical result of a cancerous out-growth in the stomach, or a venous occlusion in the liver, or as the less obviously physical result of extreme congestion inherent to the vessels or tissues of the mucous membrane, seems thus to be a main condition, a *causa causativa,* of gastric ulceration; which, in so far as it is the effect of a chemical act of solution brought about by healthy gastric secretion, constitutes a process very unlike that which we elsewhere connote by this term. The "ulcer," in short, seems to be in its outset a lesion of the vàso-motor system:[1] an accident, so to speak, of extreme gastric congestion.

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[1] See p. 38.

TREATMENT.

The means by which we endeavour to effect the cure of this disease may be all grouped together as subservient to one or other of the following purposes. Of these purposes it is not too much to premise that, just as they constitute the special indications of our treatment, so the efficacy of any particular remedies we may select will depend chiefly on the accuracy with which the latter fulfil them. To remove all local obstacles to the cicatrization of the ulcer; to support the constitution in effecting this process; to remedy the results the lesion may have already brought about; and to limit or arrest some of the more distressing and prominent symptoms by which these results are usually betrayed;—this is what we have to attempt, and can often really accomplish, towards the cure of an ulcer of the stomach.

In briefly glancing at these remedies, we will accord to medicines, as distinguished from diet, a rather unmerited precedence, and consider them first. Such an order is perhaps the more convenient, in that the mitigation of symptoms which they can certainly effect is generally the first step towards the slow progressive cure of the malady.

The question of *bleeding* is one which might almost be left unnoticed, save by a simple protest against either the general or local forms of depletion. A malady which not merely originates, but consists in, a process of destructive absorption; which, in most instances, implies some hæmorrhage, in many, a dangerous or fatal loss of blood; which is generally attended by symptoms of extenuation and cachexia; and which specially affects the poor, the intemperate, the ill-fed, the wretched, and the aged—is not one in which any presumable local benefit can counterpoise the obvious general mischief producible by even a moderate loss of blood. That a few leeches to the epigastrium will often afford relief to the gnawing pain which torments the sufferer from gastric ulcer, I see no reason to doubt. That vascularity is (to speak somewhat vaguely) so far a condition of pain, that the re-distribution of the blood which even a small general bleeding brings about may for the time diminish suffering, seems also very probable. But since depletion gives but a temporary relief, while its weakening effects on the constitution have a most mischievous local reaction, its adoption in this malady may be compared with the practice of the drunkard, who stills his stomach by a dram, which soon reproduces the very craving it has for the moment appeased.

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[1] Upon the class of lesions thus cursorily referred to, I may refer to Dr. Handfield Jones's valuable and original work just published.

But such a view of the effects of bleeding by no means applies to other forms of that revulsion which a French medical author has characteristically termed the "moiety of medicine." Blisters, turpentine stupes, mustard poultices, dry cupping, and hot fomentations, are exceedingly useful adjuncts of this kind to internal remedies. The symptom which seems to me chiefly to demand the application of such revulsives is pain; especially that continuous gnawing pain which, in severe cases, sometimes occupies even the intervals of the meals. The epigastrium is generally the most convenient site for their application. But when the chief pain is referred to the dorsal region of the spine, they are often more effective when applied to this part. As regards the particular agent to be selected, the above enumeration tolerably represents the order of their usefulness, as well as of their severity. Blisters are generally most serviceable in young and well-nourished subjects, and in comparatively recent cases, in which they are not by any means contra-indicated by pallor of countenance; the less so, indeed, that this symptom appears to be sometimes partially due to gastro-intestinal congestion. Tartar-emetic is rarely or never advisable: it is too painful and troublesome, and lowers the patient too much, even if it does not sometimes give rise to other (and more specifically antimonial) effects on the system. Turpentine and mustard, which spare even that limited expenditure of the *liquor sanguinis* implied by the serous effusion of a blister, are preferable in old and chronic cases, in which the strength is reduced; where they have the further advantage of being repeatable at shorter intervals of time than the preceding counter-irritants. Lastly, in those cases in which the powers of the system are exhausted by constant vomiting, and (what is rarely the case) the pain[1] still forms a very prominent symptom, dry cupping is the best means of mitigating it. Indeed, this derivative seems to be efficacious even in those depressed and languid states of the circulation, in which turpentine or mustard lose much of their effect.[2]

A still more valuable remedy, of an opposite kind, may be often found in the local application of cold by means of ice. In many cases of obstinate vomiting or severe pain, great relief may be obtained by frequently swallowing small lumps or raspings of pure ice. And in cases of hæmorrhage its use is almost indispensable. Here, too, it is sometimes advisable to apply it externally, by means of a bladder partially filled with powdered ice, and kept in contact with the epigastrium for a few minutes at a time.[3]

The drugs recommended for the relief of this malady are so numerous and diverse, that it is scarcely possible to avoid some classification in discussing them. But considering how moderate is our acquaintance with the action of many of them on both the healthy and diseased organ, the only justifiable approach to a classification is one that considers each with relation to those special symptoms which experience shows it can relieve. In gastric ulcer, as in many other diseases, we must both choose and classify our remedies on an empirical basis: and use their known physiological and pathological effects chiefly as means of suggesting their trial, explaining their action, testing their efficacy for the disease in general, or forbidding their application to the particular case. To accord to our limited knowledge of this kind a higher function than such a suggestive and deliberative one, would often lead us to overlook, or even refuse, excellent remedies. To rate it at a lower value would soon lead us to confound recoveries and cures; and thus to repeat the most dangerous (because the most plausible and seductive) fallacy of homœopathy.

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[1] The pain generally becomes greatly diminished on the access of exhaustion: a fact which requires no explanation.

[2] One unpleasant consequence of such applications I may mention, though it would rarely afford a definite contra-indication to their use. In two instances that have fallen under my notice, in which the necropsy revealed old adhesions between the stomach and the wall of the belly, blisters gave rise to a marked increase of the pain. A similar uncertainty attends the application of hot fomentations to the epigastrium; since in rare instances these seem to increase the pain, by raising the temperature of the subjacent parts, including the seat of the lesion.

[3] The use of external cold being strictly empirical, both in these cases generally, and in any given instance, it is hardly worth while to inquire into any imaginary danger of congestion of the ulcerated organ by revulsion of blood from the cooled abdominal surface. But how efficiently the stomach itself may be cooled by ice applied to the epigastrium, is illustrated by a converse fact;—namely, that an enema of cold water at 45° Fahr. soon lowers the temperature of the anterior wall of the belly by three or four degrees. (Compare E. H. Weber in Müller's "Archiv." 1849, Heft 4, p. 273 *et seq.*) The effect of cold substances taken into the organ is well shown by an observation of Dr. Beaumont's (*op. cit.*, p. 218), in which the ingestion of water at 55° Fahr. lowered the normal temperature of the stomach during thirty minutes.

The relief of severe and continuous *pain* is best effected by sedatives, and especially by opiates. The form in which these should be administered is determined chiefly by the state of other symptoms. Where severe pain is accompanied by very frequent vomiting, a small pill of solid opium,[1] or of the watery extract of this drug, is often better retained than any other opiate. Where diarrhœa is present, the compound kino powder is an excellent remedy; with which we may combine what is calculated to allay both the flux and the pain—the trisnitrate of bismuth.

There are probably few drugs which have enjoyed a more durable or extensive repute than this preparation of bismuth in many gastric affections attended with much pain. In the "morbid sensibility" of the older medical writers (a form of dyspepsia which, there can be no doubt, always includes a certain proportion of gastric ulcers, and which probably included a still larger number when this disease was comparatively unknown to the physician), the bismuth is constantly praised as an efficacious sedative. And my own experience not only corresponds to that of other observers, in respect to its usefulness in this variety of indigestion, but would lead me to regard it as equally beneficial in gastric ulcer. In doses of from ten to twenty grains, at intervals of six or eight hours, either alone or in combination with five to ten grains of the compound kino powder, it often has a remarkable effect in relieving the pain and vomiting so frequently present in this malady; as well as the diarrhœa sometimes combined with these symptoms.

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[1] For the other uses of opium in this malady, see pp. 174, 177, and 194.

The mention of this preparation of kino suggests some allusion to other vegetable astringents. Diarrhœa is so seldom a dangerous complication of gastric ulcer, that they will rarely be indicated in this way. And their value as tonics is greatly diminished by their tendency to cause constipation; as well as by the irritation which large doses of them in the liquid form often excite in the ulcerated stomach. They have, however, been recommended in what is called "pyrosis," with the plausible notion of checking that excessive secretion from the stomach, to which this[1] watery regurgitation or vomiting is often regarded as due.

It is not easy to give any satisfactory explanation of the operation of bismuth in these cases. To say that it is a sedative, is merely to express, by a vague generalization, its efficacy in relieving pain: and is neither more nor less correct than it would be to call it an astringent, because, in other diseases, it often limits or arrests diarrhœa. How far both these effects are akin to those of the salts of lead is difficult to determine: though its affinity to the latter in some other respects may partially suggest such a comparison.

Thus there can be little doubt that it is generally decomposed by the sulphuretted hydrogen of the alimentary canal, and converted into a black sulphuret of the metal.[2] And, in proof of this, I may mention, that its continuous administration frequently gives rise to the formation of a bluish-red line along the dental edge of the gums, analogous to (but wider and redder than) the line of sulphuret of lead in the situation which constitutes the well-known test of the presence of lead in the system. Such a de-oxidation and reduction possibly has an intimate relation to its effects as above alluded to.

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[1] Respecting the nature of pyrosis (which is by no means infrequent in gastric ulcer), see Lecture VI.

[2] A decomposition which it probably shares with the salts of lead, iron, mercury, arsenic, and many other metals.

It is not often that *flatulence* constitutes a very distressing or prominent symptom of gastric ulcer. Depending, as it does, almost solely (p. 62) on a decomposition of the food introduced into the alimentary canal, unchecked by the agency of the gastric and intestinal secretions (which normally limit and restrain all such decomposition), its presence in gastric ulcer implies a derangement of these secretions, which is not by any means necessarily (or very often) present. While a still more efficient reason for its absence is found in the fact, that any food in excess of what the quantity and quality of the various secretions can dissolve and digest, is pretty sure to give rise to an act of vomiting, by which it is soon expelled from the stomach.

We do, however, sometimes meet with cases of gastric ulcer, in which the long interval that intervenes between the meal-time and the paroxysm of pain, together with the extreme distension complained of by the patient, refer the symptoms (at least in part) to such a passive origin. In these cases the remedies usually administered for the common flatulent forms of dyspepsia appear to be very serviceable. Amongst these we may enumerate the alkaline carbonates (preferably the bicarbonate of potash), the action of which seems to depend in great degree on their neutralizing[1] the lactic and other acids developed by gastric decomposition; and the hyposulphite of soda, which appears to have a specific influence in checking that development of confervoid growths that constitutes (or, at any rate, invariably accompanies) the more complete forms of fermentation in the contents of the stomach. With the former I have found it advantageous to combine very small doses of the iodide of potassium. And of all the ordinary bitters, I have found none so beneficial, in flatulence attended with nausea or vomiting, as the infusion of calumba.

Vomiting, by far the most dangerous and important of all the symptoms of the lesion, may also be regarded as that which is, on the whole, the least amenable to the influence of drugs.

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[1] But as these acids increase the corrosive effect of the gastric juice, the benefits which attend their being neutralized are at least partially referable to its reduced solvent power.

That variety of vomiting which accompanies the painful nausea and flatulence just alluded to, may often be relieved by the remedies mentioned, especially by a combination of the infusion of calumba with the iodide of potassium and the bicarbonate of potash. Where it resists them, the hydrocyanic acid, the effervescing mixture of citrate of potash, or both these in combination, will sometimes allay it. But I have not found much benefit from creosote; and think that the salts of ammonia (in which I include the effervescing mixture of the citrate or tartrate) generally do more harm than good. Indeed, there are many cases of obstinate irritability of the stomach in which we are ultimately obliged to extend the latter statement to all the drugs generally recommended for the purpose of checking vomiting. Practically, I have long come to the conclusion, that where one or two of the above remedies appear to be inefficacious, it is better not to risk irritating or exhausting the stomach by what are really new experiments. In these terrible cases, in which the ulcerated organ is often so irritable that vomiting is produced by the smallest quantity of the blandest food, what, indeed, can we expect from the contact of unnatural and nauseous ingesta like many such drugs? Of course, if experience showed that they really did check vomiting, it would be absurd to reject them for want of a full explanation of the way in which they produced this effect. But as it may be confidently asserted that they often fail to do any good, however carefully administered, and that the treatment which foregoes their use is far more successful, it is obviously important to consider how far that treatment is more rational than the plan it claims to surpass.

That variety of vomiting which occurs in gastric ulcer could be readily shown, from any analysis of a large number of cases, to possess all the ordinary relations of this act (p. 49 *et seq.*) to the system at large—to be composed, so to speak, of all the ordinary elements of the process; to be by turns provoked by central and peripheric (or cerebral and gastric) irritation; to be favoured and opposed by the ordinary *juvantia* and *lædentia* of activity or distension, and inactivity or emptiness, of the stomach respectively.

Now it is according to these circumstances that we may best classify the vomiting present in different cases of gastric ulcer. And the same circumstances will also to a great extent dictate the means of its treatment.

For instance, the kind of vomiting most frequently seen in these cases, and that which is usually the first to appear in the history of the malady, occurs as the climax of that paroxysm of pain which begins shortly after a meal, and is referable in great part to the distension of the stomach; perhaps increased by flatulence, and aided by the stimulating effect of food and gastric juice on the raw surface of the ulcer. As such, it ends so soon as the organ has voided its contents. And it is best treated by limiting the amount of ingesta, relieving flatulence, and neutralizing undue acidity.

In the gastric ulcer of drunkards we may often notice a morning paroxysm of vomiting, which is sometimes (but not always) accompanied by pain. This vomiting, which occurs on an empty stomach, seems to be chiefly cerebral: in other words, the result of that state of general depression into which the constitution falls, as the more immediate effect of the alcoholic stimulus subsides or wears off. In exact consonance with this explanation, it is not unusual to find it occurring quite independently of gastric ulcer; while it is often habitually solaced or appeased by a dram. And of the drugs that one may more justifiably prescribe with the same object, few have greater efficacy than opium, which may be preferably given in the solid form, as a small pill of the watery extract.

The vomiting sometimes assumes a greater frequency, so as to be excited by the smallest quantity of food, or even to be repeated when the stomach is void of all alimentary contents. In many of these cases it is not easy to say whether the act is chiefly cerebral or gastric:—whether it is mainly kept up by habit, or by the debility of the patient acting on a nervous apparatus whose normal functions have been shaken or reversed; or, whether it is still, in some sense, the result of a stimulation or irritation applied to the ulcerated surface itself. In many instances, however, the associated pain points pretty conclusively to the latter view. Here it is extremely important rightly to appreciate the principles of treatment.

And, in accordance with the above suggestions, instead of ranging over the drugs to which experience or tradition attributes the power of allaying vomiting—which we may often do with little more success than if we were attempting to arrest sea-sickness by the same means—it should be our object to afford the stomach as complete and prolonged a repose as the necessities of the organism will allow. As regards drugs, the interruption to such repose, which their administration implies, will often do far more harm than their specific medicinal virtues can possibly do good. And though ice and iced water are less objectionable, yet there are some cases in which the same rule will apply to these simple and unirritating sedatives, even in the smallest quantities.

As respects food, our task is often much more difficult. There are instances in which the patient is already so much exhausted by the inanition which protracted vomiting implies, that any severe plan of diet—such as involves simple food in very small quantities, or protracted abstinence—becomes quite inadmissible. Indeed, the patient is not unfrequently in a state which would itself demand the sedulous administration of food and stimulants, if these were not contra-indicated by the state of that organ into which they must first pass.

Hence, in all cases in which there are any appearances of exhaustion, we cannot be too watchful over the effect of any system of diet we may have thought it advisable to adopt. It is, indeed, extremely difficult to gauge to a nicety how small a quantity of food will prevent inanition, and at how long intervals we may administer it. But it is well to bear in mind, not only that vomiting implies the total loss to the system of the food it rejects, as well as of fluids which are almost as valuable, but that the act itself rapidly exhausts the various nervous and muscular tissues by which it is chiefly effected. On this ground, the retention of a small quantity of food is preferable to the vomiting of a large one, even though the latter act may perhaps leave a greater surplus behind it in the stomach. And it is often important to begin by a decided plan of diet, even where the patient's weakness would necessarily forbid it being persisted in. For the habit of vomiting once so far broken through, that the stomach can tolerate a minute quantity of any food, it is frequently easy to advance to larger quantities, and better qualities, of nourishment, without provoking any return of the sickness. These points will be further alluded to in connexion with the subject of diet.

The occurrence of vomiting with dangerous frequency and intensity ought, however, always to suggest the adoption of another form of administering both food and medicines; a form which, while it concedes to the diseased organ the most perfect repose, has the merit of completely obviating the above contra-indications. I allude to the use of enemata; by means of which we may often gain a day or two of complete rest for the stomach, and thus interrupt, by an equal interval of absolute tranquillity, the hitherto habitual vomiting. Those who have had the gloomy duty of battling with all the resources of their art against the starvation gradually inflicted by obstructive cancer of the œsophagus, must have been occasionally struck by the length of time to which life can sometimes be protracted, by a sedulous adoption of this plan of nourishing the system. In spite of what seems to be perfect occlusion of the œsophagus, and even in spite of that additional extenuation which is produced by the cancerous cachexia, or by secondary cancerous deposits in the lungs or liver, the patient may sometimes be kept alive day after day—one might almost say week after week—by such alimentation only. Cases of this kind at any rate show[1] how much enemata may be made to compensate a temporary disuse of the stomach; and how safely, in obstinate vomiting, we may by their aid allow the stomach the day or two of rest, which are often all it needs. The substances best made use of are milk, raw eggs, cod-liver oil, strong unsalted beef-tea, wine, and in extreme cases even diluted brandy or opium.[2]

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[1] It is very possible that the analogy between the obstructed œsophagus and the ulcerous stomach is so far imperfect, as that the gastric mucous membrane of the former remains capable of secreting an abundance of healthy gastric juice, so long as any food whatever enters the organ; while that of the latter is often robbed of its secretory powers by the circumstances of its disease. Indeed, the symptoms of large gastric ulcers indicate at least a partial effect of this kind. But I do not think that this conjecture, even if proved to be correct, would modify the practical deduction stated above.

[2] Here, as elsewhere, I have sacrificed accuracy of arrangement to usefulness;

The adjustment of the size and frequency of these enemata, as well as their choice, must be chiefly determined by the history and the existing features of the particular case. It should, however, always be our object to reduce such enemata to the smallest possible size. And, as a rule, it is rarely advisable to repeat them at intervals of less than three or four hours. The ingestion of liquids may be best accomplished by external applications; such as wet bandages around the arms and legs, and prolonged fomentation or immersion of these limbs in tepid water.[1] With the aid of such appliances, the patient may easily be restricted for a time to the frequent moistening of his mouth and fauces with a few drops of liquid.

Hæmorrhage is another symptom which requires to be treated with especial reference to the pathology of the lesion that produces it. The scanty flux that occurs in the earlier stages of gastric ulcer—often amounting to little more than a streak or two of blood in the vomit, or a dark coloration of the stools—scarcely demands any special treatment, beyond that which it is necessary to adopt in all cases of gastric ulcer. But where it occurs in a more advanced stage of the lesion, or amounts to a considerable quantity of blood, we are justified in directing to it a more special (if not exclusive) attention. And as the stomach is amenable to the local action of the astringents we introduce into it, as well as to that general action which their absorption into the system may induce, our means of arresting hæmorrhage are (other things[2] being equal) much more efficacious than in the case of bleeding into the lungs.

The course to be generally adopted is dictated, partly by the above conditions, partly by the existing state of the patient. In respect to the latter, I would suggest (at the risk of again incurring the suspicion of leaning too much towards an expectant mode of treatment) that where there is reason to suppose that

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and have overstepped the not very definite boundaries of food, stimulants, and drugs. In speaking of the above remedies, however, I am for the time regarding both of them as stimulants; which, with diverse rapidity and certainty, may not only arouse the flagging powers of the system, but may economize (and therefore substitute) a certain amount of food.

[1] To which milk may be sometimes added with great advantage.

[2] For some of the qualifying circumstances hinted at by this phrase, see pp. 60 and 121.

the bleeding comes from a large vessel, and is for the time arrested, it is better to limit our treatment to the negative plan of securing for the stomach the most perfect rest. A little ice from time to time, if the patient be very thirsty, or complain of much pain; a rigid observance of the supine posture; and the minimum of food (preferably of boiled milk) that will support life:—such are the simplest and best precautions we can take, to avoid the disturbance of that clot which alone intervenes between the life and death of the patient during the first few days that immediately follow a copious hæmorrhage from a gastric ulcer. A more frequent and moderate oozing justifies (and demands) a styptic treatment, especially where there is no great tendency to vomiting present. Turpentine, and the sesquichloride of iron, which have, I believe, been recommended with this view, are both open to the grave objection of often exciting nausea and vomiting, even in moderate doses and dilution. The formula I most prefer consists of about ten grains of gallic acid, dissolved in an ounce of distilled water by the aid of about ten minims of the dilute sulphuric acid. But in some cases of this kind, in which the presence of great pain has led me to prescribe bismuth and compound kino powder, the bleeding has seemed to be arrested by these astringent drugs.

Even here, however, the internal and external application of cold by means of ice, and the rigid observance of the dietary already referred to, forms an indispensable part of the treatment.

The *cachexia* which generally accompanies the gastric ulcer, and the hæmorrhage which, in varying amount, usually occurs in some part of its course, unite to constitute one of the most important indications of treatment. This cachexia, which combines and expresses the worst effects of all the other symptoms, and which constitutes (I am convinced) one of the most frequent causes of death in the general history of the malady, not only modifies and limits our application of the remedies hitherto specified, but practically measures the general success of our treatment.

The difficulty it sometimes opposes to that local treatment by which the ulcer is best allowed to heal, has already been alluded to. The system of stimulation it sometimes dictates, we shall again revert to in speaking of the dietetic treatment of the malady. At present we may chiefly view it as indicating the exhibition of *tonics* in all cases in which the state of the ulcer does not absolutely forbid the introduction of these remedies into the stomach.

Amongst the various remedies included in this class, the preparations of iron claim the foremost rank. The pathology of the chlorotic condition which is simulated by the gastric ulcer of the young female, as well as the results of the hæmorrhage often implied by the lesion, alike point to these preparations; which are the best means of furthering such a growth of the red corpuscles of the blood, as may obviate that anæmia, or repair this hæmorrhage. And experience quite confirms these rational expectations of the efficacy of iron.

The chief precautions necessary to observe in its use seem to be the following. Iron is contra-indicated by frequent vomiting, and by excessive or continuous pain. And even when these symptoms have partially yielded to other remedies, it is better to begin with the very mildest preparations of the metal, such as the ammonio-citrate, or ammonio-tartrate. They should always be given either with, or immediately after, food;—a general rule in using them, which the presence of an open ulcer makes doubly important. And the various soluble salts of the metal should be prescribed in combinations which allow them to be introduced into the stomach in this dissolved form. In other words, the use of the insoluble oxide, either directly or indirectly (as in the compound iron mixture a few hours after being made up), should generally be avoided.

The bitter vegetable tonics are, on the whole, less important; though, in combination with other remedies, they are often useful. Thus the infusion of calumba is not contra-indicated by any moderate amount of vomiting; and that of quassia may often be given with iron. Lastly, of all the ordinary combinations of mineral and vegetable tonics, none is so elegant and so generally useful, in the later stages of convalescence from gastric ulcer, as a mixture of the sulphates of quina and iron, kept in solution by a few drops of dilute sulphuric or hydrochloric acid. Indeed, wherever the stomach will bear quina (as it certainly will in the majority of cases after suitable preparation), a course of this energetic tonic, during at least a few weeks, is very advisable. And in those instances in which the ulcer itself seems to be a sequela of ague, such a rule becomes, for obvious reasons, still more valid.

Another combination of quinine and iron with opium will often be found of great advantage for irritable stomachs; and may be prescribed with the best effect in cases where either of these drugs in the liquid form would give rise to much inconvenience—such as nausea, vomiting, headache, or loss of appetite. It consists of a pill of about a grain of each of the above sulphates, with a small quantity of the extract of opium, or the compound styrax or soap pill. The marked advantage of such a pill over the above mixture is probably due to its slower solution, and to the comparatively smaller gastric surface on which its direct action is exercised.

The use of *aperients* in this malady requires the greatest caution; although, in many cases, they can hardly be dispensed with. It is impossible to doubt that they have sometimes been administered in this and other gastric diseases far too indiscriminately; and that their abuse has occasionally led to very prejudicial results. How far they are really necessary in any given case, is, however, a question which it will often be extremely difficult to decide. Still we may recollect, that where little food is taken into the stomach, or much rejected from it, the bowels often contain so small a quantity of excrement, that the need of defæcation becomes far less frequent and urgent—to say nothing of that abdominal stagnation (compare p. 121) which the mere act of vomiting itself tends to produce. On the other hand, there can be no doubt that constipation may seriously increase both the pain and vomiting of gastric ulcer. Indeed, in one or two cases, I have noticed a definite and repeated coincidence between paroxysms of these symptoms, and an accumulation of fæces in the colon. But, not to speak of the clue the statements of the patient will generally afford us, a careful examination of the belly will almost always assure us whether the large intestine is occupied by even a moderate amount of fæces.

When an aperient is definitely indicated by appearances of this kind, few remedies answer better than castor-oil. A moderate dose (from six drachms to one ounce) of this purgative can often be taken, in the ordinary way, without at all increasing the pain or vomiting. But where the latter symptom is of frequent occurrence, it is much better to resort to enemata, either of castor-oil in emulsion, or of some combination of this drug, and the decoction of aloes, with the gruel which is usually the vehicle of such remedies. As a rule, large enemata are not advisable; distension of the abdomen being very liable to increase the gastric pain and uneasiness.

The milder aperients sometimes required during convalescence may be given under limitations similar to those already mentioned with respect to the stronger purgatives. When their use can be avoided, it is better to omit them altogether. Indeed, on the subsidence of the more serious symptoms, the state of the bowels often affords a fair test of the efficacy with which the tonics administered are acting upon the system. In any case, an occasional dose of the compound rhubarb pill, mixed (if need be) with an equal quantity of the compound extract of colocynth, is quite sufficient; or a dinner pill of one or two grains of the extract of aloes may be given daily, with the same object. Of course, the slightest symptom of relapsing pain or nausea would quite contra-indicate such remedies. And a proper study of the patient's diet will often enable us altogether to dispense with the use of aperient drugs.

These brief allusions to aperients naturally suggest some mention of the *mercurial* preparations which so often enter into their composition. It would be a dereliction of duty in me not to express the strong opinion I entertain against the employment of mercury in these cases, in any form, and under any pretence whatever. I believe that I have known one or two instances in which the ulcer has been definitely produced by the administration of mercury for other maladies; and am certain that I have witnessed relapses which could only be attributed to a similar cause. A single calomel purgative has even appeared to undo all that months of sedulous treatment had been able to effect towards the relief of a gastric ulcer.

With a different feeling, I venture to allude to some of the remedies, the efficacy of which has been greatly extolled in this malady. The preparations of *silver*—and especially the oxide and the nitrate—have been alleged to possess a therapeutical effect of the most marked kind. Indeed, it is suggested by some of the advocates of the nitrate of silver, that it heals an ulcer of the stomach just as it would an ulcer of the leg; that it stimulates the languid atonic ulcer to a new action, and soon brings about a process of granulation.

It would be presumptuous in me to question the excellent effects of this caustic in the external ulcers to which surgeons often apply it; though I suspect benefits are often attributed to the stimulus of its application, which depend chiefly on that coagulum or crust of organic matter which it forms, and by which it so admirably shields and compresses the subjacent granulating surface. But, in respect to its internal administration, I have no hesitation in saying that, as ordinarily prescribed, it is absolutely inert; that the benefits which have been observed under its use, are due either to the diet and adjuvant remedies also adopted, or to the curative efforts of Nature; in short, that the above estimate of its value really reflects and repeats the fallacy—*post hoc, ergo propter hoc*—which lies at the basis of the fancied cures vaunted by homœopathy.

In specifying the facts on which I ground this opinion, I limit myself to the doses and combinations in which it is generally prescribed.

The constitutional effects produced by this or any other salt of silver, when absorbed into the system, I may altogether eliminate from the question. Without going so far as to deny that these salts have any constitutional action, I may at least assert, that none such has ever yet been proved; that they "may be cautiously exhibited for a considerable period without producing any obvious changes in the corporeal functions;"[1] and that the only evidence adducible to prove their influence over the constitution, consists in the rare amelioration of epilepsy, or the still rarer appearance of a cutaneous eruption, under their use. And, apart from this absurdly defective evidence, we may question whether any such action results from their ordinary use. For that blue colour of the skin which testifies to their absorption and reduction throughout the body is so specific an event, that, in its absence, we have no right to assume any definite series of constitutional effects whatever.[1] While, since the most zealous partisans of these drugs carefully stop short of producing this unsightly and permanent change of colour, we are justified in supposing, that whatever effect their doses have, is exclusively due to a local action of the remedy on the mucous membrane to which it is applied.

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[1] Pereira's "Materia Medica," vol. i. p. 692.

Now, as regards any such local action, it is of little consequence whether the usual half-grain or grain dose of nitrate of silver is administered in the form of a pill or of a solution. Far from its giving rise to any symptoms of reaction or irritation, such as we can see (and the patient feel) when we apply any form of this caustic to an external or internal surface, the pain and irritation, the feverishness, nausea, or vomiting, which have been sometimes noticed to result from the remedy, are expressly regarded as indicating an over-dose. In fact, all evidence of direct stimulation as a result of the remedy would probably be interpreted as proof that it had been wrongly administered.

And just as the absence of all such evidence of the stimulant effect of the drug leaves us in very great doubt whether its known physiological action is really exerted on the gastric membrane, so its equally well-known chemical reactions convert this doubt into an absolute denial.

We will say nothing of the chlorides of the food, the chlorides of the gastric juice, the chlorides of the bread-crumb in which the drug is often exhibited. Suppose all these sources of decomposition carefully avoided—a supposition, however, which is really untenable—the chlorides of the saliva and œsophageal mucus swallowed with the pill, and of that mixture of these secretions with bile which occupies the fasting stomach, would suffice (and more than suffice) to convert any ordinary dose of nitrate or oxide of silver into this insoluble compound. And what chance a tiny pill has of seeking and finding in the stomach a resting-place which exactly corresponds to the ulcer;

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[1] Thus the constitutional effects of lead are rarely unaccompanied, and mostly preceded, by its characteristic blue line around the gums.

or what chance the minute dose, diluted by an ounce of liquid, has of being uniformly diffused over the gastric surface of about 180 square inches, and yet of super-saturating the mucus of the same place;—it would be absurd seriously to inquire.

I need scarcely say that, in urging these[1] objections to the efficacy of the oxide or nitrate of silver in this malady, nothing is further from my intention than to deny that patients have ever recovered under their administration. But, considering that we may allege the same fact of almost every curable malady, under the administration of every "panacea" with which quackery has ever afflicted mankind, few would be inclined to lay much stress on it. Indeed, its explanation is sufficiently obvious. Pathological research conclusively shows that ulcers of the stomach often undergo a spontaneous cure. The careful system of diet which is usually adopted in conjunction with the remedy, is itself sufficient to account for all the benefits observed. And I am persuaded that the physician who judges of the efficacy of his remedies without allowing for the natural tendency of disease to recovery, and for the effect of proper diet and regimen, while he shrinks from testing their traditional virtues by a careful scrutiny of their physiological effect in the healthy subject, and their therapeutic[1] influence apart from diet—that such an one, I say, has little to prevent him from exchanging one system of credulity for another, and enlisting under the banner of any the most abject quackery of the day.

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[1] While referring to Lecture VI. for some further incidental remarks on the evidence which decides the therapeutical efficacy of drugs, I venture to deny that the arguments on which Heller and others come to a conclusion, apparently resembling the above, respecting the general inertness of this salt of silver, are applicable to the question of its efficacy in gastric diseases. Even as respects its constitutional action—considering that it is an absence of proof, rather than any disproof, of absorption, which chemistry affords us—our ignorance of the minimum of this and many other metallic salts requisite for such constitutional action, refers us to analogy, and to physiological and pathological considerations, for evidence for or against its operancy. And as regards the usefulness of the nitrate of silver in gastric maladies, the mere discovery of chloride of silver in the fæces is a piece of evidence both needless and inconclusive. Needless; for surely it needs no analytical chemist to tell us what happens when a soluble chloride is added to this nitrate. Inconclusive; because the supposed benefits of the drug are plausibly referred to its local effect;—an effect which is no more disproved by the discovery of the insoluble chloride in the secretions of the stomach, than in those of the vagina, after its application to the mucous membrane of these two organs respectively. And since (as the latter analogy shows) the general reaction of the local effect of this caustic may amount to a constitutional effect of a most important kind, it is rather a question of contact, than of absorption, with which we are here concerned. While it must be remembered, that the same overstrained argument from analysis would oblige us to deny the unquestionable efficacy of bismuth in many gastric and intestinal maladies.

Diet.—The comparative importance of this element of treatment has already been alluded to. So far as I can judge, it is impossible to cure ulcer of the stomach by any remedies, in the absence of proper regimen. It is probable, that in many of those cicatrized ulcers which have been verified by a necropsy, the patients' own feelings had guided them to such a diet as might fairly be considered the agent of their recovery. It is quite certain that many cases have been cured by the observance of a rigid system of food, which has been prescribed to the exclusion of all drugs.[2] Hence, though the latter are (in my opinion) invaluable as aids to a strict diet, they are powerless as substitutes for it. And in no disease is it therefore more important for the physician to protest against that common error of the unprofessional public, which looks upon the deglutition of a certain quantity of more or less nauseous drugs as the only means requisite for the restoration of health.

The system of diet which experience shows to be the best treatment for this malady is based upon certain simple and well-ascertained principles in the physiology of digestion; such as have but to be mentioned, to be at once admitted as unquestionable. The sufferers from this disease find their symptoms greatly aggravated by copious meals; by animal food; by hard, tough, and indigestible substances; by hot food or drink; and lastly, by irritant substances. While, conversely, the use of a bland, moderately-azotized food, of soft, pulpy consistence, at a cool temperature, and in small quantities, not only relieves the symptoms, but often, if steadily persevered in, completely cures the malady. And every one of these circumstances precisely corresponds to what the physiology of digestion, and the pathology of the lesion, might, *a priori*, teach us to expect. The organ which the ulcer occupies is stimulated to activity by the mere ingestion of food. It is stretched and distended by a large meal. And just as the ulcer necessarily shares in these changes, so its raw surface must be stimulated by the heat, and by any irritant qualities (mechanical or physiological) which the food may possess. Lastly, as it is the express function of the stomach to dissolve the proteinous constituent of the food, the amount of this constituent present in the diet adopted will not only to some extent measure the degree and duration of the activity of the organ, but will probably also affect the ulcer more specially, by regulating the quantity of gastric juice secreted into that general cavity of the organ which the lesion occupies.

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[1] Such a therapeutical influence—a matter of immediate and frequent experience—as is illustrated, under just these circumstances, by bismuth, alkaline carbonates, and many of the so-called "carminatives."

[2] The case of Béclard, the French anatomist, seems to have been an example of this kind. He died at an advanced age, after a long period of perfect health; and the scars of two ulcers were found in his stomach.

The various requirements implied in the preceding statements are best fulfilled by milk diet, given in small quantities, and at frequent intervals. This fluid, the natural food of the young animal, and the model alimentary mixture of the scientific cook, contains all the substances necessary for the organism, not only in suitable proportions to each other, but in a state which specially favours their digestion, and involves no mechanical irritation of the stomach. The quantity of protein, for example, is such, that there is little risk (as in the case of some of the simplest and blandest varieties of vegetable food) of our depriving the organism (perhaps already exhausted by disease or age) of its necessary supply of this indispensable constituent of food. While, conversely, it probably does not much exceed the proportion required by the wants of the system.

It is chiefly by the irritability of the stomach (as expressed by the frequency and facility of the vomiting) that we must be guided in our selection of this or that modification of milk diet. Where the stomach is excessively irritable, the milk may be sometimes better borne when diluted with half or one-third its bulk of lime water, or after a preliminary boiling. In rare instances, a little fresh curd, mixed with a thin pulp of arrow-root boiled in water, seems preferable even to these. As a rule, however, good new-milk is far more easily sustained than either of these modifications; and the chief efforts to adjust it to the stomach are to be made by carefully regulating its quantity.

Where the more moderate irritability of the stomach allows a better chance of food being retained by the organ, it is very important to give articles of diet which possess a greater physical consistence. This is best effected by mixing with milk any of the purer varieties of starch, and boiling them together into a thin pulp, which of course must only be taken when cooled. Arrow-root is especially to be recommended for this purpose. Sago and tapioca are generally less easily borne. The gradations of quantity and frequency must also be carefully prescribed for the patient. The advantage of taking a single spoonful of such food for a meal consists in the facts, that not only is all distension of the stomach thus avoided, but also that the food is thus enabled to leave the stomach shortly after entering it—becoming merely impregnated, as it were, with gastric juice, before it passes on into the duodenum, to receive its complete solution there.[1]

The frequency of such meals must of course be regulated by their quantity, as well as by their effect on the stomach. Thus, supposing them not to excite vomiting, from ten to twenty meals of a single table-spoonful or dessert-spoonful of the above articles of food will not be too many during the whole day—a number which would obviously demand their being repeated as frequently as every two hours.

As convalescence begins and proceeds, it is important to keep up as rapid an advance in the scale of diet as prudence will allow. We may at first vary the pure starch of arrow-root by passing to sago and tapioca, before venturing on the moderately proteinous ground-rice, to advance thence to maize, rye, or wheat-flour. Ground-rice I have generally found well borne; and both in respect to consistence and composition, it forms, with milk, an excellent staple food, on which many persons can live without risk for days or weeks together. Wheat-flour is best given in the shape of biscuit-powder, or bread steeped in boiling water, and pressed through a muslin sieve while still hot (bread jelly), boiled with milk. Sugar must be be added with great caution, as it is very apt to provoke flatulence.

It is chiefly when, from such a diet, we desire to advance to ordinary animal food, that the greatest difficulty will be generally felt:—a difficulty which a foreigner unacquainted with the wasteful and barbarous processes by which the average Englishman cooks his food could scarcely appreciate. Strong beef-tea is rarely well borne: and boiled or roast meat is often too sudden a change; indeed, if badly chosen or cooked, it may do serious mischief. It is my usual practice to begin with some boiled white fish (such as sole or brill) carefully freed of all bones, and mixed with mashed potatoes or water arrow-root in small quantity. But a tender meat very slowly stewed, so as to form what the French call "bouilli," is almost as good; though far less likely to be obtained by most patients. Of course it would be quite possible for a patient to subsist on vegetable food with only the aid of milk. But so few persons can be trusted (or expected) permanently to isolate their habits from those around them, that we have practically no choice but to arrange for their return to the ordinary diet of health. There are many other articles of food (the suggestion of which belongs rather to cookery than to physic) that must be taken with the same precautions, as to quantity and consistence, which we have already mentioned for the milk diet.

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[1] See pp. 3, 140; also *Cycl. Anat.*, *op. cit.*, pp. 315, 349, 398.

The idiosyncrasy of the patient often obliges us greatly to modify any such plan as the preceding. Sometimes water may be advantageously added to the milk; though it cannot be long substituted for the more nutritious fluid, unless something of more azotized composition than arrow-root be given with it. Some patients can soon take an egg beaten up with their farinaceous food; while in others it evidently aggravates the previously improved symptoms. Some take cold tea with advantage; although, as a rule, this fluid ought to be strictly prohibited. In short, the diet—nay more, the cookery—in each case requires careful observation, as well as great prudence and tact. Every change is to be regarded as in some degree an experiment, the carefully observed results of which can alone justify or forbid its continuance.

Stimulants.—The question of employing stimulants naturally claims our notice in connexion with diet; although we have already briefly alluded to them in speaking of the drugs sometimes indicated by that exhaustion or cachexia which, in varying amount, it is usual for gastric ulcer to produce. As a rule, it is indispensable to avoid all use of alcoholic stimulants. Even in the extreme exhaustion produced by protracted and frequent vomiting, it is generally advisable so far to keep to this rule, as to administer whatever alcohol seems necessary in shape of an enema.

The rare exceptions to this rule are rather to be hinted at as contingencies than stated as facts. In respect to absolute experience, I have certainly known one or two glasses of sherry, and even one or two tumblers of beer, to be taken daily, without much appearing to impede the cure of what seemed to be an ulcer of the stomach. But I do not think the comparatively slight mischief which the disobedience of these patients produced is at all incompatible with the rule above laid down. So many of the middle-aged subjects of this lesion are persons of intemperate habits, and so much does the lesion itself increase the depression and cachexia which these habits produce, that it is quite possible the constitutional advantages of a moderate amount of stimulus may sometimes exceed (in other words, more than neutralize) the bad effects of that local irritation which the raw surface of the stomach is sure to suffer from its application.

Of course, if any form of alcohol were admissible, it would be a pure dilute fluid (such as weak cold brandy and water) in very small and frequent doses. Raw spirits, and brandied or strong wines (such as Marsala and sherry respectively) are inadmissible, from their irritant effects. And such compound or artificial drinks as ordinary port, beer, and champagne, are still more objectionable; their sugary or fermenting constituents generally aggravating the injurious effect of their alcohol to a remarkable extent. The effect of the true or natural wines in small quantities I am hardly qualified to speak of; though, from one or two instances, I am entitled to believe them much less hurtful.

Opium.—This antagonism between the local and constitutional effects of alcoholic drinks in the treatment of gastric ulcer, may well suggest the inquiry, whether there are no stimulants which would afford us the advantages of alcohol without its disadvantages? The importance of this question will excuse my pointing out, that what I have already had occasion to say respecting the merits of opium will, to some extent, apply to the class of sedatives in general. But the peculiar stimulant effects of opium make it by far the most valuable of them all.

Respecting the operation of this drug in gastric ulcer, I might say much that would be both interesting and true, but nothing that would include those elements of accurate physiological and chemical research on which alone a conscientious physician ought to base a definite theory of the action of this (or any other) medicine. As regards mere facts, I am quite certain that, though the pain often present in these cases is of course an additional indication for the use of opium, yet it is by no means the chief (far less the only) guide to its administration. It is not as an anodyne, not even as a sedative, that opium seems to be most useful. On the contrary, my experience would lead me to conclude, that it is especially in ulcers of long duration, of large size, of obstinate character, and in broken exhausted constitutions, that this invaluable remedy comes more fully into play;—and that the condition these circumstances presuppose being present, its use is not one whit less advantageous, even though the habitual pain is but trifling, or though (far from having to replace the customary alcohol of the drunkard) it is prescribed for a patient who has been always of a temperate or even abstemious habit.

In short, I am anxious specially to urge upon you the importance of giving opium in this dangerous and frequent disease, with just the same views as those with which I suspect it has long been employed in phthisis, or (a still more apt illustration) in diabetes. To relieve this or that pain; to check great irritation, or undue secretion, of this or that mucous membrane, supposing such symptoms to be present. But to support the strength, to buoy up the nervous system, and to check the waste or expenditure of the tissues generally, whether the above local symptoms are present or no. That it is thus opium aids to heal a gastric ulcer, I can scarcely doubt. And that, in this way, the same (I would even say, the only[1]) drug which considerably diminishes the saccharine constituent of a diabetic patient's urine, may also check that process of destruction which a gastric ulcer expresses and measures, is quite in accordance with all that we know respecting its power of lessening bodily waste.

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[1] See the author's monograph already cited, p. 144.

But I am more anxious to state the fact of its usefulness in combating this lesion, than to explain the principle upon which it does so. It is, therefore, with very great pleasure that I have found my own experience of its efficacy in ulcer of the stomach confirmed by the statements of other observers with respect to its uses in ulcer of other parts of the body. You may take the analogy of the two classes of ulcerative lesion at what it appears to be worth; and may estimate, at a higher or lower rate, that remarkable parallelism which the pathological details I have established respecting gastric ulcer exhibit with those of the ordinary ulcer of the leg.[1] But should you be disposed to think favourably of statements founded on such wide research as mine have been, you will probably hardly fail to be struck by their perfect agreement with the independent results of a better authority in the treatment of a kindred disease. Allowing for the situation of the lesion, and for a variety of details which that situation chiefly dictates, there is hardly any difference between the opinion I have come to respecting the efficacy of opium in ulcer of the stomach, and that which has been published by Mr. Skey,[2] as deduced by him from his large hospital practice, with respect to its benefits in ulcers of the limbs.

As regards the mode of administering opium, where vomiting is moderate, or where diarrhœa is prominent, the compound kino powder is a very convenient formula. But when vomiting is at all excessive, or resists a combination of this powder with bismuth, the drug is generally better borne in the solid form; either as a small pill of the watery extract, or a few grains of the compound soap or styrax pill, two or three times a day. The effect of opium given in this way often seems to be quite as striking as in ordinary ulcers of the leg.

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[1] As shown by Mr. Critchett's able treatise, already (p. 163) referred to.

[2] Report of Clinical Lecture, the "Lancet," vol. i., p. 88, 1856.

For the same aged and intemperate classes of patients in which opium is so generally beneficial, other sedatives will occasionally afford similar (though less marked) results. Hyoscyamus, conium, belladonna, and other extracts of this kind, are thus sometimes beneficial, though I have rarely had occasion to resort to them. The extract of hemp sometimes answers admirably; indeed, were its effects more uniform, it might often be advantageously substituted for opium itself.

In all cases, the return of the convalescent subject of gastric ulcer to his ordinary diet should by no means end his precautions with respect to the disease. Many months after a complete recovery, I have repeatedly known a single excess bring back the whole train of symptoms, with all their attendant dangers. It is therefore only after the lapse of a long period of perfect health that we can venture on suspending the system of diet now described. Indeed, no lapse of time justifies a person who has once suffered from this disease in laying aside many of the above rules. The subject of this malady, whether convalescent or cured, ought, in fact, always to retain the habit of taking food in small quantities; making up, if need be, for their smallness by a greater frequency of meals. His food ought not only to be well chosen, and good of its kind, but well cooked, and carefully masticated; so as to secure its reaching the stomach in a proper pulpy consistence. While hot food and drink, as well as alcoholic excesses, ought to be systematically avoided.

We may end these suggestions by repeating two precautions respecting the mechanical treatment of gastric ulcer; precautions which ought never to be neglected.

Firstly, as regards its situation, it is very important to avoid all pressure on the epigastrium, especially where there is much tenderness of this region to such interference. The injurious effect of tight stays, or of any hard substance (such as the end of the last used by shoemakers), is of course obvious enough; though I have reason to believe such pressure may rupture a stomach without its habitual application having previously produced much pain. But in the examination which the physician has to make (and which is sometimes imitated afterwards by the patient), we are ourselves bound to remember this injurious effect of pressure; and to be not only very gentle in our manipulations, but very sparing in our repetition of them.

Secondly, all violent exercise must be avoided. In cases where life was threatened by hæmatemesis, or by constant vomiting, few would forget to adopt this precaution. Indeed, the feelings of most patients would soon enforce the recumbent attitude, even if the medical attendant did not prescribe absolute rest in the supine posture. But in persons who merely regard their complaint as an aggravated indigestion, it becomes more necessary to state such a rule;—a rule, the infraction of which may be punished at a moment's notice by the terrible accident of perforation, and is almost always attended with both local and constitutional results in the form of pain, vomiting, fatigue, or exhaustion.

LECTURE IV.

Cancer of the Stomach.

The gastric maladies we have hitherto successively studied have, as it were, gradually risen in severity to find their climax in cancer of the stomach ;—a disease which, obscure in its symptoms, frequent in its occurrence, fatal in its event, will, on all these grounds, well repay that careful clinical study, of which I shall briefly describe what seem the most important results.

Let me premise, that I can not only offer you some new details bearing on the diagnosis and treatment of the disease, but may claim an accuracy scarcely hitherto attainable. Indeed, the researches on gastric ulcer epitomized in the last Lecture themselves suffice to demand a revision of the whole subject of gastric cancer. For they show, not only that the frequency and importance of the former disease have been underrated, but that its symptoms and appearances, though clearly distinguished twenty years ago by the sagacity of Cruveilhier, are yet very liable to be confounded with those of the cancerous disease it can so closely simulate. Now it is evident that the study of gastric cancer in general, as well as its differential diagnosis from gastric ulcer, requires the accurate distinction of these two diseases from each other. And nothing short of such a discrimination having been made would justify my calling your attention to the original observations, and the large series of necropsies, which the following Lecture sums up and incorporates.

The typical course of the malady may be sketched as follows.

An elderly person, perhaps hitherto free from dyspepsia, begins to suffer from a capricious, and soon a diminished, appetite ; which is by and by associated with occasional nausea or even vomiting, and with a sense of uneasiness or distension in the stomach. His complexion, already pale and unwholesome, next acquires a muddy yellowish, or faint greenish hue. His gastric symptoms now increase; often by a sudden and marked augmentation, which corresponds to what is, in other cases, their first appearance. Vomiting, if already present, becomes more frequent or urgent; local uneasiness deepens into pain: and both these symptoms are excited or increased by taking food. At a somewhat later period, hæmorrhage generally occurs; usually but scanty in amount, and therefore depending to a great extent on casual circumstances for its detection. About this time a tumour often becomes perceptible near the middle of the epigastric region of the belly. As the local symptoms increase, the cachexia of the patient also augments; and is evidenced, not only by the colour already mentioned, but also by debility and emaciation: and at last by prostration, which ends in anasarca, delirium, and death.

The disease, however, often diverges from this course: the above symptoms being complicated in various stages of their progress by results more or less incidental to the locality of the cancer—such as ascites, jaundice, perforation, fistula, phlebitis—or by the addition of the various symptoms characteristic of consecutive cancerous deposit in other organs, especially in the liver and lungs. Most of these complications influence the termination of the malady, either as inter-current or as concurrent causes of death. And, in any case, the disease generally marches to its termination with a continual increase of speed and severity, rarely receiving more than a temporary check, and ending in death about one year after it first declares itself.

In discussing each of the chief symptoms enumerated above, and in noticing some questions connected with their succession, combination, and duration, it will be convenient here and there to anticipate some pathological considerations, which either explain these symptoms, or are suggested by them. Theory in any wider sense it is my express object to avoid: not that I consider it useless or unimportant, but rather that I doubt whether it can be advantageously entered upon in the present state of our information, and especially in these Lectures.

Loss of appetite seems, on the whole, the most variable and vague among the leading symptoms of gastric cancer. Numerically, it is present in the great majority of cases; or, more exactly, in about 85 per cent. of their total number.

But it is not in all these cases that the anorexia forms an early and marked symptom of the malady. On the contrary, its appearance is often deferred until a comparatively late period. And its intensity is still more frequently restricted to a degree which allows little stress to be laid upon it, apart from the circumstances which attend the disease. In rare instances, too, its variations amount to absolute intermissions. And in still rarer cases, the appetite is increased, instead of diminished, by the malady.

The value of this symptom as an element of diagnosis is therefore considerably less than might be supposed from its numerical frequency. Its presence is of little influence in this respect, unless it is early, as well as prominent, in the history of the case; unless it precedes by some time any violent or continuous pain or vomiting; and unless it is accompanied (rather than followed) by cachexia and debility. Where it exhibits these features, it combines with the other symptoms as a valuable piece of evidence, and is especially useful in assisting to distinguish between cancer and ulcer. For in the latter of these two maladies, loss of appetite is so rare, that we may almost deny its claim to be considered a symptom at all. True, the subject of gastric ulcer often evinces a great repugnance for food. But a close inquiry would show that this repugnance rarely depends on anorexia. The patient is afraid to eat, simply because experience has taught him the suffering (pain and vomiting) which this act excites. Indeed, except in the later stages of the malady, the cravings of his appetite are (for obvious reasons) rather increased than diminished; so that, every now and then, instinct prevails over judgment, and he eats a hearty meal. In cancer, on the contrary, anorexia often seems to be a specific result of the disease. As such, it appears to be effected through the same nervous[1] channels which mediate the ordinary sensations of hunger and satiety, and to commence during a very early stage of the deposit in the coats of the stomach, prior to any more local symptoms. It is, on the whole, more marked in the younger subjects of the disease, and in the softer varieties of cancerous deposit.

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[1] In my experience, a sudden loss of appetite, and change of colour, have frequently

The sex of the patient appears to exercise no influence whatever on the presence or absence of anorexia in the history of the case.[1]

Pain, the next symptom, is on the whole more frequent and specific than anorexia.

It appears to be present in about 92 per cent.: in round numbers, in nearly 11 out of every 12 cases. The sex of the patient seems little to affect the painfulness of the malady. And there is scarcely more reason to suppose that the situation of the lesion in the stomach has any such influence.

The region to which the pain is referred affords little deduction as to the exact site of the lesion, at any rate in its earlier stages. Thus a pyloric cancer may not only cause pain referable to the right hypochondrium, or to the epigastrium, but even to the umbilicus, the sternum, or the left hypochondrium. In cancer of the cardia, again, the pain may not only be local, but may be referred to either hypochondrium, or even to the right shoulder. The cancer of the lesser curvature seems, however, to be sometimes connected with a peculiar reference of the pain to the inter-scapular region, as well as with a remarkable intensity of the pain. And any considerable involvement of the posterior surface of the stomach

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preceded all other symptoms. But the most curious form (?) of such anorexia I ever witnessed was in the case of a German gentleman, whose ordinarily slender appetite was little affected, but who suddenly found himself indifferent to tobacco, which he had smoked largely, though not to excess, throughout his life. He was a fine, handsome man, of exceedingly long-lived ancestry. His medical attendant regarded his state with profound suspicion; but could verify nothing but a deterioration of colour and appearance, and the change above referred to. After a careful examination, I satisfied myself of the absence of every other sign or symptom of disease. Nevertheless, as a matter of professional instinct, I could only confirm and define the suspicion of my colleague, by diagnosing gastric cancer. In the course of a few weeks the case became only too distinct; and in a few months the patient died of an unmistakeable cancerous stomach.

[1] For the details which amplify this and other statements of the same kind, as well as for the facts on which they are based, the reader is referred to an essay "On Cancer of the Stomach," in the "British and Foreign Medico-Chirurgical Review," 1857, p. 475, *et seq.*

often causes a pain which ranges from the middle of the dorsal to the lower part of the lumbar region.

But a careful inquiry into the import of this symptom soon resolves numerical statements like the above into their more trustworthy (though less definite) constituents. The pain of gastric cancer is evidently no single and comparable element of its history in all cases—like the pain present in most instances of gastric ulcer—but is rather a variable (or even complex) symptom, producible by several circumstances, which impart to it a very different relationship to the malady, according as they are its exclusive or predominant causes in any given instance.

The pain most characteristic of the malady is usually of a lancinating character. Beginning at a comparatively early date, it rapidly assumes a marked severity; often becoming so intense in the course of but a few days as to cause the patient an amount of suffering which throws every other symptom into the background. Its fluctuations scarcely ever amount to more than remissions: in other words, it rarely intermits, or ceases, for any length of time. Unlike the pain of gastric ulcer, it is either little affected by the ingestion of food; or, if increased by eating, does not subside at the end of the act of gastric digestion, and after vomiting—of which act it is often quite independent. As time goes on, such pain sometimes becomes less urgent, subsides, or even, in rare instances, disappears. More frequently, however, it changes its character; merging, in greater or less degree, into some of the following varieties.

In many cases, indeed, the pain is from the first devoid of any distinctly lancinating character: and is variously described by different patients as dull, slow, gnawing, or burning; and as being attended by a sense of weight, oppression, tightness, or distension in the epigastrium; or by soreness or tenderness to pressure in this region.

Many of these varieties of pain appear due to the local circumstances of the cancerous lesion: and indeed, may often be traced to specific causes, such as go far to explain, not only their characters, but even the way in which they sometimes complicate or succeed each other in the history of any given case. The dull burning variety of pain seems to belong rather to ulceration of the cancerous stomach than to the cancerous deposit itself; and as such, often closely simulates the pain of gastric ulcer. Thus pain of this kind is increased by taking food, is referred (it may be) to an epigastric and a rachidian spot, and is relieved by the expulsion of the gastric contents. At the same time, its accession to the symptoms of the malady sometimes seems to relieve the more specifically cancerous pain—perhaps by that mechanical relief of tension which ulceration and hæmorrhage (its constant companions) together imply. Local tenderness or soreness is also referable to ulceration; though, where excessive, they allow a presumption of that adhesive inflammation so common in both cancer and ulcer of the stomach. A sensation of weight appears but rarely to correspond with a really heavy or bulky tumour. Tightness, oppression, and distension (often connected with weight) frequently belong to that constriction of the stomach which the cancerous deposit brings about: and when best marked, have repeatedly been observed in conjunction with a maximum of "stenosis;" with or without dilatation of the gastric cavity behind the seat of stricture.

Hence the circumstances of these varieties of pain assign them a comparatively subordinate relation to the malady. Its various stages imply events which themselves conditionate various abnormal sensations; such as we must, for the sake of pathological accuracy, eliminate from any inquiry into the pain produced by the cancerous deposit itself. Subtracting these incidental pains, we are left with a symptom which, though greatly reduced in mere numerical frequency, has a more characteristic aspect, and therefore a more direct bearing on the nature and diagnosis of the disease.

Specific pain of this kind is probably not present in more than one-half the total number of gastric cancers. And it undoubtedly varies, not only within the limits which would ascribe it a lancinating character of greater or less intensity, but even in the fact of its possessing this character at all. It may be dull, aching, or even burning; may remit or even intermit; may appear late, or vanish early in the history of the case.

Many would probably consider that such facts went far to show a subjective character of the pain:—that they exceeded, rather than fell short of, the evidence upon which some good authorities regard as subjective the pain of malignant tumours occupying parts governed by the cerebro-spinal system of nerves.

It is because I believe this view to be not only erroneous, but likely to exercise an injurious influence on pathology, that I venture to point out how baseless are the assumptions on which it rests. Equivocal as is the language of logic when applied to the physiology of innervation, still it allows us to perceive that such a view is placed in the dilemma of being either untrue in its facts, or, if we correct these, still more faulty in its deductions.

For it is the very essence of pain not to be objective:—in other words, not to be precise or accurate in the information it gives of the properties of any object. Even when brought about by the application of a stimulus to the peripheric organization of a nerve, it gives little or no intelligence respecting the stimulus itself; so that, for example, a red-hot poker, and a few drops of liquid carbonic acid, would give rise to much the same kind of pain when applied to the palm of the hand. And when the stimulus by which pain is produced impinges on the nerve at some point between its periphery and its centre, the pain becomes even more subjective; gradually losing all appreciation of the locality affected; and referring (for instance) an injury of the ulnar nerve at the elbow, to its distribution in the fingers.

Hence, to the statement that—"the pain of malignant tumours is subjective," it might fairly be answered, "True: but what pain is not so? On the other hand, if by 'subjective' you mean subjective in any other sense than the pain of a wound or a burn; or if you regard the uncertain and changeable relations between the malignant lesion and the pain it produces, as showing that there cannot possibly be the same physical chain of causation as that which obtains when a nerve is involved in such injuries—then you are implying a statement which centuries of pathological research may perhaps hereafter gradually build up, but which is at present a mere assertion. Not until we know immeasurably more of the structure and function of nerve, and of the exact relations of the cancerous deposit to the terminations of the nerves among which it lies, shall we be entitled to say that the adventitious mass does not provoke pain by a process precisely similar to that which may be set up by a mechanical or chemical lesion or injury. In the meantime, to remove pain into the region of the 'subjective,' is to withdraw it from those very researches by which alone its claims to this position can ever be substantiated."

But to regard any such theory as merely "not proven," is to take a far more favourable view of it than is warranted by facts. The nature of the above varieties of pain, and the accuracy with which they generally correspond to the various conditions mentioned, conclusively refute it. So also does the study of the pain more specific to gastric cancer. As in malignant tumours of other parts, the maximum and minimum of pain in cancer of the stomach can generally be traced to something more than a conjectural cause. Excruciating agony has two or three times been found to coincide with a definite involvement of some large branches of the pneumogastric nerve in the cancerous deposit. And conversely, the presence of little or no pain has far more frequently been concurrent with such a general hardening of the whole organ, as has converted the stomach into a rigid inflexible tube, by a scanty but diffuse deposit involving scarcely any displacement of its tissues. The somewhat greater pain of the disease in young subjects appears to be connected with their greater proneness to the softer and more rapidly developed forms of cancer, and with the greater local injury[1] which these usually imply. Indeed, allowing for the indirectness of the connexion between the stomach and the cerebro-spinal system,[2] the pain produced by gastric cancer seems to be rather less irregular than that produced by malignant tumours elsewhere.

Vomiting seems to be scarcely less frequent than pain in the history of gastric cancer; being probably present in $87\frac{1}{9}$ per cent. of its cases.

There is no sufficient ground for deducing any influence of sex on the presence or absence of this symptom. Apparently it is rather more frequent in the gastric cancer of the female, in whom the proportion of 87 per cent. witnessed in the male, rises to $89\frac{2}{3}$. Or, to put the fact more strikingly, the absence of vomiting throughout the malady is nearly one-third (1·3 to 1) more frequent in the male. But the numbers concerned are too small to justify any stress being laid on so limited a difference. Indeed, even if substantiated hereafter, it will probably turn out to be chiefly connected with the collateral circumstances of this symptom—for instance, with its occasional late appearance, allowing it now and then to be (as it were) anticipated by general exhaustion.

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[1] Under opposite circumstances, even enormous fungoid growths may be quite painless. Compare "British Medical Journal," 1857, p. 493.

[2] A connexion, the influence of which may be roughly estimated by comparing the pain of cancers without and within the chest.

The locality occupied by the cancer appears to affect the occurrence of vomiting in a much more marked degree; although the number of examples I have collected only justify such a conclusion in the case of the more frequent situations of the disease. From an analysis of 167 instances which specify the necessary details, it would appear that this symptom favours cancer of the different parts of the stomach in the following order of increasing frequency: the posterior wall, the whole organ, the middle, the lesser curvature, the greater curvature, the cardia, the pylorus.

Since the numbers in the lesser curvature and in the two latter localities together make up five-sixths of the whole,[1] their comparison is more trustworthy. They especially show (what, indeed, has long been conjectured) that cancer of the pylorus is more frequently attended with vomiting than cancer of any other part of the stomach; and suggest that this symptom is sometimes brought about chiefly by obstruction of the gastric cavity—that is, by such obstruction as any deposit of cancer around the narrow calibre of the pylorus would be likely to produce.

A further study of this symptom indicates that it may be conditionated by three or four different causes; the precise share of which in the vomiting of any particular case it is sometimes impossible to estimate.

Firstly, there seems to be a variety of vomiting (as of pain) which is at any rate so far specific or inherent to the malady, as that it is producible by a scanty deposit in the coats of the organ, and is therefore observable at a very early period in the history of the disease. The frequency of this form of vomiting cannot be determined. But I should conjecture it not to occur in more than ten per cent. of the total number of cases; and to be little (if at all) more frequent in cancer of the stomach, than in non-malignant deposits in or on this part of the digestive tube. It seems to be connected chiefly with a local irritation of the nerves distributed to the seat of the disease; and hence to vary chiefly with the abruptness of outline of the deposit, and with the displacement it inflicts.

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[1] See the remarks on the pathology of this lesion in a subsequent part of this Lecture.

Next in the history of the disease, and of greater numerical frequency, comes the vomiting produced by obstruction or by stenosis. It is affected by locality, as above mentioned. And in rare instances it is relieved or removed by the ulceration or sloughing which destroys the obstructive part of the cancerous mass.

A third variety of vomiting appears to form a still larger fraction of the above total frequency of its occurrence. That process of softening and ulceration which the cancerous deposit sooner or later undergoes, first removes the mucous membrane, and then erodes a variable extent of the original or adventitious tissues beneath; thus giving rise to an abnormal irritation which rarely fails to cause vomiting. In general, the symptom, once thus excited, recurs more or less frequently throughout the further course of the disease; a fact sufficiently explained by the continual increase of the lesion, and by the influence of habit on the act. Occasionally, however, it subsides or disappears; sometimes it would seem, from the low vitality or irritability of that softened or ulcerous mass which comes to form the inner surface of the stomach; oftener (for a similar reason) from the access of general prostration.

Lastly, there is a less frequent (but more complex) class of cases, in which this symptom is apparently due to a failure of contraction in the muscular coat of the stomach, giving rise to a virtual obstruction which itself provokes vomiting. In rare instances, this interruption to peristalsis may be due to a nervous cause, such as would justify its being termed paralysis. More frequently it is traceable to a direct interference with the muscular structure; a large extent of which is removed by ulceration, or weakened by dilatation, or starved (and even destroyed) by interstitial deposit.[1]

Each of the above four varieties exhibits peculiarities such as often aid to distinguish it. The first, for example, generally forms the climax of a gradual increase of anorexia and nausea; and rarely shows any marked relation, either to the quality of the food, or to the period of digestion. The second offers a close resemblance to the vomiting seen in gastric ulcer; and, like it, is provoked by food—especially by large meals, and by hot, irritating, and proteinous articles of diet.[2] As a rule, however, it is less constant, less intense, and therefore less dangerous, than the vomiting of gastric ulcer; and is often limited to the last few weeks of the patient's life. The third generally occurs at intervals of many hours—indeed, is usually a large (from two to six pints or more) dark yeasty-looking vomit, which is the accumulation of many meals, and contains *sarcinæ, torulæ,* or both: while it is of course accompanied with the physical signs of dilatation of the stomach. The fourth can only be said to afford direct and independent evidence of its presence in those rare (but authentic) instances in which the patient has felt that the contracted stomach could neither receive more than a small quantity of food (all in excess of this amount being instantly vomited), nor pass onwards any appreciable fraction of it into the duodenum.

Hæmorrhage constitutes a less frequent symptom than either of the preceding: being noticed in about 42 per cent. of the cases on record:—a proportion which is the same in both sexes.

In about one-sixth of these instances, the hæmorrhage was sufficiently copious to be recognised as tolerably pure blood. The remainder include a great many cases in which the blood appears to have been more or less mixed with gastric juice, food, bile, or softened cancerous substance; and to have undergone a variable change of colour, such as would impart to it a blackish, brownish, or "coffee-grounds" appearance.

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[1] It is true that the evidence afforded by cases of gastric cancer rarely amounts to more than a presumption that the vomiting present is chiefly or exclusively caused by one or other of these forms of interference with peristalsis. But it must be remembered that the efficiency of every one of them is proved by its constituting the sole cause of this symptom in some other diseases of the stomach. (See Lecture V.)

[2] With reference to the share probably taken by the gastric juice in producing the ulceration which causes this vomiting, compare pp. 163-5.

But the frequency of hæmorrhage is doubtless much greater than the above statements suggest. Indeed, the pathology of cancer in general, together with the history of this particular malady, will quite entitle us to suppose that some hæmorrhage rarely fails to occur, save in those instances in which the patient dies before the ulceration or softening of the deposit. But though, in all other cases, its occurrence seems necessary or inevitable, its detection is contingent; being determined by a variety of collateral (or even remote) circumstances,—such as the quantity of blood poured out, the rapidity of its effusion, the casual coincidence of vomiting or purging, or the attention habitually bestowed by the physician or patient upon the substances thus expelled. And hence there can be little doubt, both that a sedulous inquiry (aided if necessary by the microscope) would reveal this symptom in many cases in which it must otherwise remain unnoticed, and that the above numerical statement greatly underrates its true frequency.[1]

In respect to their nature and origin, such hæmorrhages may be divided into three varieties. First in the history of the malady comes the hæmorrhage of congestion; which often occurs at an early stage of the deposit; and which, though usually moderate in amount, is in rare instances so copious as to cause death. In these fatal cases, the accuracy of the term congestion is rendered probable by the fact, that a careful examination of the stomach, aided by injection of its vessels, conclusively shows that the hæmorrhage has proceeded from the minute vessels of the sub-mucous plexuses, or from the capillaries of the free gastric surface, and not from any of the trunks or larger branches of the arteries or veins. In other instances, the relation of the tumour to such a congestion has been still more definitely indicated by a varicose enlargement of some of the small veins in its neighbourhood, or a deep purple streaky coloration of more or less of its periphery. Whether the passive or obstructive congestion which such a state suggests is more or less frequent than an active congestion or afflux to the adventitious growth; and whether the latter state, in so far as it occurs, may not sometimes diminish, rather than aggravate, the former;—are questions which cannot at present be answered. Thus much we may, however, conclude respecting this variety of hæmorrhage:—that while its very occurrence in gastric ulcer is in some degree hypothetical,[1] it appears to form a definite and not infrequent form of bleeding in gastric cancer; and seems to be often produced by a passive or physical engorgement of the vessels, itself the result of a mechanical obstruction brought about by the deposit.[2]

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[1] Compare p. 119.

The second variety of hæmorrhage is the result of the various lesions in which the processes of ulceration and softening involve the vessels of the cancerous mass. In conformity with this source, it generally occurs at a comparatively late period of the malady, and is by far the most frequent cause of the more copious bleedings witnessed in cancer of the stomach. The degeneration and destruction by which it is produced are often complicated by a process of fungation or out-growth, which increases the amount of blood effused by such hæmorrhage. As a rule its amount is generally scanty—often sufficiently so to escape detection. In rare instances, however, it is excessive; and from the circumstances that attend it, is always important (often fatal) even when but moderate in quantity.

The third variety of hæmorrhage is that produced by erosion of the larger vessels external to the stomach. Its production and comparative infrequency will be again alluded to.[3] Unlike the two preceding varieties, hæmorrhage of this kind is probably but seldom overlooked. Hence the estimate that it does not cause death in more than one out of 100 cases of gastric cancer, or in more than one of the 40 of this 100 which evince such a symptom, probably does not understate its relative frequency. From obvious reasons, it generally occurs at a late period of the disease, as measured both by its duration, and by the succession of its symptoms.

The characters of the blood expelled from the stomach of course vary with all those circumstances which affect the appearance of this fluid in the analogous hæmorrhage that attends gastric ulcer. According as it is poured out by an artery, or a vein; in gushes, or by a slow oozing; in large, or small quantity; and is expelled at once, or only after undergoing more or less admixture and digestion—the blood detected in the patient's stool or vomit may be liquid or clotted; florid, dark, or black; pure, or so changed, and combined with other contents of the alimentary canal, as almost to defy recognition.

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[1] See pp. 119, 151. The coincidence of cirrhosis of the liver with ulcer of the stomach is suggestive, however, of such hæmorrhage (p. 165).

[2] Future observations can alone show whether villous cancer affecting the stomach is attended with that peculiar disposition to hæmorrhage of this kind which it seems to exhibit when occupying other mucous membranes.

[3] See the pathological section of this Lecture.

As regards the relative frequency of its two means of exit, hæmorrhage seems to be detected solely in the matters vomited, about five times as frequently as in the stools exclusively. In one-fourth of the cases, it is found in both these evacuations. Such proportions correspond to five-eighths, one-eighth, and two-eighths respectively.

It is scarcely necessary to say that the so-called "coffee-grounds" vomiting, which was formerly supposed to be almost pathognomonic of cancer of the stomach, has really no such diagnostic value. It occurs in gastric[1] ulcer; as well as in various other lesions of the organ attended with scanty extravasation of blood. And its greater frequency in cancer is attributable rather to the moderate amount in which hæmorrhage usually occurs in this disease, than to any more specific cause. At any rate, this circumstance, and the comparatively greater frequency of vomiting on an empty stomach in cancer than in ulcer, seem quite to explain the disproportion observed.

In close connexion with hæmorrhage is another symptom, till now almost overlooked—namely, the detection of the characteristic cancerous cell-growth in the substances expelled from the stomach.

For obvious reasons, this sign is rarely to be found, except at such a date of the malady as would generally imply the presence of most (if not all) of the preceding symptoms. Indeed, depending (as it does) rather upon exfoliation or sloughing, than upon any mere slow dissolution, of the cancerous mass, it is intimately associated with such a degeneration or decay of its substance as we have already seen can rarely occur without more or less hæmorrhage. And although it is quite possible that sedulous research might detect cancerous matter in the stools of a patient, still, in practice, there is scarcely any reasonable chance of its discovery, save in those cases of gastric cancer in which vomiting is not only present, but occurs upon an empty stomach, and with tolerable frequency.

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[1] Melanosis is so extremely rare as a cause of this colour, that the diagnosis of its nature is identical in the two diseases. Compare p. 120.

The ordinary difficulties in the recognition of cancer under the microscope need scarcely be alluded to; although these difficulties are increased by the circumstances of its deposit in the stomach—especially by its form (almost always medullary at its exfoliating surface), and by the solvent effects of the gastric juice. For however the cancerous nature of the substances expelled may be obscured by the equivocal size and shape of their cells, and by their separation and dissolution in the stomach, its recognition is practically much more impeded by the large and numerous impurities admixed with them here. The addition of blood, pus, bile, saliva, and gastric juice, together with the various and complex admixture of particles entering into the composition of the simplest foods and drinks, will often frustrate the most careful and repeated examination of these egesta.

The only practical rules I can offer are the following. The fluids selected for examination should be such as are vomited tolerably free from any admixture of food. If much blood or bile be present, they should be diluted carefully by the addition of a moderate quantity of water. Thus attenuated, they should be searched (by decanting off the clear fluid) for any flocculi or larger masses they may contain. If the examination of these be at all delayed, syrup will form the best means of preserving them. To the practised eye there is little danger of confounding a mass of cancer, even when already half dissolved, with shreds of œsophageal or gastric epithelium. But mere isolated cells or nuclei scarcely justify a decision.

On the whole, it is evident that this symptom, rarely visible until little doubt is left, often defying all detection, sometimes yielding but equivocal evidence, can only now and then be of value. And its usefulness is solely positive. In other words, its absence proves nothing. In both these respects it stands in marked contrast with the analogous evidence afforded by the microscope in some other diseases:—especially (for example) in suppurating pulmonary tubercle; where the distinctness of the tissue detected, and the comparative constancy of its presence, render it a ready and unmistakeable piece of positive evidence, and with proper precautions, allow its absence some value as a negative.

The detection of a *tumour* constitutes a symptom which may be estimated as present in about 80 per cent. of the cases of gastric cancer. The sex of the patient does not seem to influence the frequency or distinctness of this symptom.

The form of cancer scarcely seems to exert any more perceptible influence on the presence of a tumour. From obvious reasons, the tumour produced by scirrhus generally appears at a comparatively late period in the history of the malady.

The situation of the cancer in the stomach seems to exercise the influence we might expect on the appearance of a tumour. Whether we calculate this influence directly (by comparing the cases of its presence and absence) or indirectly (by comparing the former cases with the general frequency of the symptom as deduced above), we obtain results which may be briefly summed up as follows. The presence of a tumour is favoured by the disease being situated in the middle of the stomach, in its greater curvature, in the whole organ, and in the pylorus; its average frequency, as represented by 100, being increased to 300, 220, 160, 120, for these four situations respectively. On the other hand, it is opposed by the cancer being situated at the lesser curvature, the cardiac, and the posterior surface; the same average being respectively diminished to 95, 44, and something considerably less than 25. These facts are obviously explained by the relative proximity of the corresponding parts[1] of the stomach to the yielding anterior wall of the belly, by contact with which an ordinary cancerous tumour of the organ is rendered easier of detection.

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[1] We may distinguish the effect of these situations by alluding to the circumstances through which they oppose the appearance of a tumour, as comprising at least three elements—(1) remoteness from the surface, as in all these sites; (2) influence on perforation, and incidental death, as in cancer of the posterior surface (see pathological section); and (3) specific tendency to grow in another direction, as in the cardiac cancer, which generally shoots up the œsophagus.

The situation of the tumour in the belly varies in a degree which is but partially explained by its anatomical relations. As might be expected, it is almost limited to the epigastric, umbilical, and hypochondriac regions; where it forms a projection of variable size, shape, and smoothness, from a large, hard, irregular, nodulated mass, causing a prominence which it is impossible to overlook, to a small, deep, elastic, or unresisting substance, such as is often exceedingly difficult to recognise. Tumours of the great curvature are apt to project near the umbilicus; cancer of the whole stomach projects chiefly into the epigastrium; the upper part of which region (from equally obvious reasons) generally shows the swelling of a cancer situated in the lesser curvature. But the variableness in the situation of the tumour is best seen in the pyloric cases, which form so large a majority of the whole. In most of these, the swelling occupies the neighbourhood of the median line. Where it transgresses this rule, it selects the right hypochondrium more frequently than the left—a preference which is referable, not only to the normal situation of the pylorus, but also to the frequency of adhesion between the diseased segment of the stomach and the contiguous liver. The vertical level of these pyloric tumours appears to be affected by the sex of the patient in a curious (though by no means inexplicable) manner. The horizontal line which separates the epigastric from the umbilical regions divides the locality usually occupied by such swellings into two pretty equal portions. Of these the upper (including the epigastric and hypochondriac regions) is, in the male, the seat of two-thirds of these tumours; the lower of one-third. The female subject of cancerous pylorus exactly reverses these proportions, two out of every three tumours occupying the umbilical regions. This strange contrast must be ascribed, partly to the naturally narrower limits of the female epigastrium, partly (and much more) to the constriction exercised by stays; two circumstances which concur to force the liver and stomach, together with any tumours which may be attached to them, to a lower level in the belly than they would otherwise occupy. The same circumstances appear to be traceable in the fact, that mobility of the tumour is much more frequent in the female—at least, mobility in such a degree as allows the tumour any wide deviations in the abdominal region it occupies.

The detection of a tumour is sometimes opposed by great difficulties. The resistance offered by the supposed mass now and then leaves us in doubt; merging (as it does by various gradations) into a slight fulness to palpation, or dulness to percussion. Again, the tumour is often in absolute contact with the liver; and hence, if of moderate dimensions, may be so completely covered by this organ, as to have its physical signs fused into those of the lobe of liver by which it is overlapped. Nay, more, a large proportion of these tumours adhere to the liver: while a still larger proportion[1] are associated with a secondary cancerous deposit in the hepatic substance, such as often defies the greatest skill to distinguish from the swelling produced by the gastric cancer apposed to it. On the other hand, the adhesion and inflammation around an ordinary gastric ulcer may give rise to a perceptible tumour, which must sometimes be suspected as cancerous. Further, the tumour produced by a gastric cancer may be temporarily hidden by ascites, as well as by changes in the size and situation of the neighbouring intestines. And lastly, just as the cancerous deposit is sometimes too scanty, and its situation too deep, to permit the appearance of this symptom at all, so I have often seen the tumour producible by a cancerous stomach closely imitated by cancer of the neighbouring mesentery or intestine. The gastric distension brought about by constriction and dilatation behind it can, however, almost always be distinguished from a cancerous tumour. Even when (as is not uncommon) the two states are combined, the presence and degree of dilatation may be decided by a physical examination; the results of which, again, can be checked or verified by the characters of the vomiting present in such cases. (Compare Lecture V.)

The above variations are well illustrated by the fluctuations in distinctness which such tumours sometimes exhibit in the course of a single case. The swelling distinguishable one day has disappeared on the next; to re-appear, it may be, a few days later. Occasionally, its subsidence is permanent: a fact which, if unexplained by any of the circumstances above mentioned, points to a loss of substance in the diseased mass, by softening, ulceration, or sloughing.

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[1] See the pathological section of this Lecture.

Many of these tumours are pulsating—in other words, are so placed as to receive and transmit the impulse of the aorta. Of such pulsating tumours a disproportionate number seem to affect the pyloric situation, and the male sex.

Mobility of the cancerous tumour seems to be rare, in that marked degree which permits it to occupy different abdominal regions. The three or four cases I have collected are all pyloric, female, and (at least originally) scirrhous.

Tenderness of the tumour is generally present in some slight degree. It is marked in about half the cases; but is rarely great, save where the peritoneum is either inflamed, or is involved in the original or secondary cancerous deposit.

The *cachectic appearance* upon which so much stress is deservedly laid in the diagnosis of gastric cancer, is scarcely a single symptom, but rather a congeries of symptoms; some of the slighter and less direct of which can neither be satisfactorily grouped with it, nor separated from it.

In respect to its frequency, about 3 in 4 of the cases on record have exhibited it in a marked degree. This proportion is exactly alike for both sexes. But in a lesser extent it is probably present in not less than 90 to 98 per cent. of cancers of the stomach.

As elements of this cachectic aspect, we may fairly enumerate, not only the colour of the skin of the face and body, but the loss of cutaneous moisture and elasticity, the diminution of the sub-cutaneous fat, the flabbiness (if I may use such a word) of the areolar tissue, and the softness and smallness of the muscles. The latter symptoms are closely connected with emaciation and debility, and often with kindred characters of the pulse and the cardiac action.

Of all these appearances, however, the altered facial colour is the most obvious and important, as well as the most constant. The more characteristic variety of this change is describable as imparting to the face a kind of muddy greenish pallor, or pale earthy hue, such as can scarcely be mistaken for any other colour. The chief extremes (if we may so term them) of this symptomatic colour are jaundice on the one hand, and an anæmic whiteness on the other.

Jaundice appears to occur in a proportion of about 5½ per cent. It may be conjectured that in about one half of these cases the jaundice is due to the hepatic disorganization produced by consecutive cancer of the liver.

In the remainder the jaundice seems quite independent of any structural hepatic disease. The coloration of such jaundice appears to merge, by numerous gradations in different cases, into the ordinary hue of the cancerous cachexia. And (if we except those rare cases in which its symptoms, and the date of its appearance, refer it to a cause analogous to that of the jaundice which sometimes comes on in the later stages of mortal cardiac disease) it is difficult to avoid regarding it as little more than a variety of this cachexia. Such a view is especially confirmed by one or two important features it generally offers. The icteroid stain is rarely intense, and even the conjunctiva is often but moderately yellow. The stools, too, however offensive, have neither the white colour, nor the putrefying odour, characteristic of true jaundice. And the skin and urine are not loaded with bile in anything like the usual degree.[1]

Pallor of the skin is just as evidently a variety of the characteristic colour in some cases, a complication of it in others. In other words, it often seems traceable to no other cause than the cachexia which it accompanies; oftener, however, is due to some of those incidents of the malady which, when they occur in other diseases, bring about a similar result. Thus hæmorrhage, suppuration, frequent vomiting, scanty ingestion of food, long confinement, and a host of circumstances (among which are recorded such rare contingencies as concurrent albuminuria) may all complicate and increase that pallor, which is an element of the peculiar facial hue characteristic of gastric cancer.

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[1] A full discussion of this interesting point would diverge too widely from the present subject. But without wishing to deny the analogy of this coloration to certain forms of jaundice, I am anxious to insist on its connection with the cancerous cachexia, and its distinctness from the commoner varieties of icterus. And while its presence in any particular case may help to distinguish it as cancer, its complete absence in others scarcely counts for much less. As regards the urine, indeed, I am inclined to consider that the condition imparted by the cachexia, attending cancer of the stomach, ranges, by all possible degrees of coloration, from a large proportion of biliverdin, through lessening quantities of this colouring matter, to the purple urates noticed by Prout, and hence down to a mere excess of ordinary urohæmatin. Practically, in any doubtful case, a normal hue and amount of urinary colouring matter is a fact of the most gratifying import.

With allusions equally hasty and imperfect, we must pass by the interesting subject of inquiry which the nature of this cachexia might afford us. Enough to say that of all the provisional theories which suggest themselves, that regarding the original or pathognomonic cachexia as the result of a humoral disease, which precedes and brings about the cancerous deposit, is, on the whole, the safest, as well as the most useful. According to such a vague theory, there is a cachexia which, in some cases, can be verified prior to any of those local changes that afterwards rudely measure and express it. But besides this specific cachexia (the nature and date of which render it so important for pathology and diagnosis), there are a variety of conditions which closely resemble it, but bear to the total malady a relation of coincidence rather than of identity, and of effect rather than of cause. Often as these conditions complicate and obscure the true cancerous cachexia, pathological accuracy requires their separation in the history of the malady; nay, more, so far as may be practicable, in any given instance. Indeed, it would be difficult to find a better illustration of the practical value of such accuracy than we may see in many cases of this malady. Contrary to what has been generally stated by authors, the cachectic aspect witnessed in gastric cancer is often imitated by mere ulcer of the stomach—sometimes so closely as really to defy distinction. But in all these cases, the resemblance, however close, is easily explained. The circumstances of both diseases are such as to involve, in a large proportion of their more fully developed cases, a certain amount of cachexia;—which is the joint product of ulceration, hæmorrhage, vomiting, pain, and starvation. While in other cases a knowledge of this fact renders the symptom a valuable aid to diagnosis. Wherever cachexia either precedes these circumstances, or is present in a degree utterly disproportionate to what their aggregate influence might lead us to expect, there it acquires the rank of a leading (and almost pathognomonic) symptom.

Febrile symptoms accompany gastric cancer much more frequently than is generally supposed. But I cannot offer you any numerical statement respecting the frequency of their presence. As a rule, these symptoms rarely amount to definite hectic, but are generally limited to what is termed irritative or symptomatic fever. The tongue, often covered with a tolerably thick white fur, which is especially distinct posteriorly, gleams through this covering, or appears at its edges of a deep-red "raw" colour. The face is not unfrequently marked by injected red patches on the centre of each cheek, contrasting strongly with the pallid cachectic hue of the surrounding integuments. The urine is still more frequently of scanty amount, high specific gravity, and abnormal colour (comp. p. 211); and is loaded with urea and urates. The various forms of uric acid deposit are also anything but uncommon; indeed, it is perhaps in this malady that we may find the best illustrations of that peculiar pink variety (or rather complication) of uric acid deposit, which has long been regarded as characteristic of constitutional malignant disease.

The import of this febrile reaction is probably very different in different cases. Ulceration, inflammation, and peritonitis, suggest themselves as its causes, scarcely less frequently than does the primary malady, or even the exhaustion it directly or indirectly produces. On the whole, however, the latter state seems to me the best clue to the nature of these symptoms in the majority of cases in which they are present. Especially is this explanation applicable where none of the above local complications appear to be prominent. In such instances, the febrile reaction often seems strictly analogous to that feverish condition producible by starvation, exposure, or over-exertion, in persons otherwise healthy. And in consonance with this view, I think it will generally be found that its presence is a rough test of the progress of the malady—at any rate, of its effect upon the constitution; and therefore not only threatens the speedy prostration of the patient, but affords an indication for as much nourishment and support as the local circumstances of the malady will allow.

The *state of the bowels* in gastric cancer is another point on which I have no numerical details to offer. In most instances, however, either constipation or diarrhœa is specified as having been present during a considerable period of the malady. And in many, the two states alternate with each other; constipation in the earlier months of the history being succeeded by diarrhœa towards the last few weeks of life.

Constipation, the more frequent of the two, appears to be often due to the circumstances of the malady rather than to the malady itself: and, in this respect, is comparable with the constipation which so frequently accompanies gastric ulcer. In many cases, indeed, the gastric obstruction, vomiting, and pain—which prevent alike the distension, and the movements, of the intestinal canal—concur to form a ready explanation of the frequency and intensity of this symptom, which somtimes persists up to the last hour of life.

But in other instances, constipation assumes a much closer relation to the diagnosis. Wherever it is unexplained by association with the foregoing circumstances (in other words, when it stands almost alone with anorexia and cachexia) it acquires significance. A sudden change in the action of the bowels—which, from regular and punctual movements every day, become, in the course of a week or two, languid, sluggish, and finally downright obstinate, yielding to cathartics, only to relapse into constipation as soon as these are suspended—becomes, in such instances, quite an element of the diagnosis. True, it does not of itself point out the affected viscus, much less the affected segment of the digestive canal. And still less does it specify the lesion. But it is nevertheless of great practical importance: even if it be not (as it sometimes has been in my own practice) recognisable by those indefinable features which, as is the case with other common symptoms, stamp this variety of constipation with the physiognomy of gastric cancer.

Diarrhœa seems mainly attributable to the direct irritation caused by the cancerous, purulent, or hæmorrhagic effusion which the gastric lesion introduces into the intestines. The noxious influences of these fluids are often aided by their decomposition in their passage through the bowels. Ulceration of the cæcum, and of the neighbouring ileum and colon, is comparatively so rare, as to afford but a very exceptional explanation. The date of the occurrence of diarrhœa is generally consonant with the above view of its production; being subsequent to ulceration—in other words, comparatively late in the history of the malady. It thus coincides with a period, at which the patient's strength is generally so far exhausted, as to be very susceptible of any further depressing influence. Hence it often indicates imminent danger, and is not unfrequently the proximate cause of death.

The symptoms which more specifically betray the approach of death, offer little that is peculiar to this disease. Anasarca is by no means uncommon, and is usually accompanied by ascites. A disproportionate effusion into the belly, points rather to some local cause of the dropsy, such as pressure of the cancerous tumour on the *vena portæ;* and is hence less significant for prognosis. Jaundice, as before mentioned, appears to be sometimes a precursor of death, as is also a form of pulmonary effusion which I believe to be mere passive congestion. Hiccough, again, is usually due to local causes acting on the diaphragm. The nervous phenomena which usher in the close of the malady are delirium, followed by coma.

The *duration* of the aggregate malady formed by the succession of all these symptoms, I should estimate, from 198 cases, as averaging about $12\frac{1}{2}$ months. This average seems to differ little in the two sexes. Out of 142 cases, 107 male and 35 female, the average in the male was $12\frac{1}{3}$, in the female $11\frac{2}{7}$ months. This slight difference is rendered even less important by the smaller number of females referred to; especially in conjunction with the fact, that the deviations from the average are far more striking, and less trustworthy, in the case of the longer durations, than of the shorter. Indeed, as regards the longest or most chronic instances, it is curious to notice how few gradations exist between protraction to the close of the second year, and the rare examples of a duration of six or seven years. Hence, it is probable that many of these extremely protracted cases are examples of dyspepsia ending in cancer, an opinion which may also be directly deduced from their symptoms.

Practically, then, we may deduce,—that the maximum duration of gastric cancer amounts to a period of about thirty-six months from the first appearance of the symptoms;[1]

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[1] Without wishing to modify the above rules, I may notice some striking ex-

and that very few cases survive the twenty-fourth month. The minimum is about one month. As might be inferred from the historical details hereafter alluded to, many of the most protracted cases are shown by necropsy to have a structure often designated "colloid" and "encephaloid," though originally scirrhus. The converse exception is much rarer, a large majority of the gastric cancers which are rapidly fatal really belonging to these two intense forms of the malady.

But in noticing these facts, we must be careful not to overrate their significance. The commencement of the symptoms often has little connexion with the commencement of the disease. Pathologically, it is of course not only impossible to fix the date of the deposit in any given instance, but probable that its material origin may be followed by all those varieties in the rate of subsequent growths which we can verify in cancers of external parts. And even that aggregate of symptoms which, for diagnostic purposes, we must regard as the representative of the disease, will often give a very distorted view of the real duration of the case. Slight anorexia, cachexia, or emaciation might often elude the notice of the patient; or, perhaps, would rarely cause him to seek professional advice, such as might sometimes detect and distinguish the latent causes of these early symptoms. Hence, in most of the cases on which the above estimate is founded, the symptoms from which the commencement of the malady has been dated are those of active gastric disturbance, in the shape of pain and vomiting.

This brings us to a point on which I am anxious to be explicit, in order not only to expose the probable bias of the above estimate, but also to explain the deceptive resemblance between cancer and ulcer of the stomach;—a resemblance which it is often of vital importance for the physician to unmask. In many cases of gastric cancer, the

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ceptions which have convinced me, after full consideration, that the pathological connection below hinted at does really obtain in many cancers of the stomach, and even of the pylorus. In some, symptoms absolutely identical with those immediately preceding death (pain, vomiting, &c.) have occurred, intermittently, for ten, fifteen, seventeen, twenty years before. In others, the first and last attack has been of but a few weeks' duration. In both classes, the necropsy has shown the whole or part of the cancer to be so shrunken, hard, or even stony, as to refer its origin to a date at least many months prior to the last illness.

symptoms only date from the commencement of ulceration in the cancerous deposit;—from a time, that is, when the incurable and malignant disease acquires a close analogy, in many of its circumstances, to the curable ulcer; an analogy such as necessarily brings about the closest similarity in many of its local symptoms. Thus, whether we explain their access as due to this cause, or view them as independent of it, it is certain that the more marked and frequent varieties of vomiting and pain, as well as of hæmorrhage, begin at a period about midway in the history of the best marked cases. Hence we may conjecture (1st) that the cancerous deposit itself generally precedes the symptoms by a period of at least some months: and (2nd) that the above estimate of the total duration of the symptoms may perhaps receive some extension from future clinical researches.

And now let me add a few words respecting the combined effect of all the preceding symptoms on the diagnosis of cancer of the stomach.

The numerical estimates I have offered are deduced from the collation of about 250 cases, verified by necropsy. But in the records thus used, each of these symptoms occupies one of three categories: it is present, or absent, or unmentioned. Now, of the six foregoing estimates, all but those relating to hæmorrhage and cachexia completely ignore the latter category; and only contrast the number of cases in which the particular symptom was present, with those in which it is as distinctly stated to have been absent. But since it may be presumed that, in many such records, it has been unnoticed, because really absent, it may be a question whether the corresponding estimates ought not to be reduced: and to a degree somewhere between the numbers we have assumed, and the subtraction (of 38, 5, 10, and 26 per cent. respectively) required on the supposition that the symptom unmentioned was always absent. On merely numerical grounds, I should, however, prefer the larger estimates; not only because they are based upon a more direct and positive comparison; but because they seem better to balance the two kinds of error—excess and deficiency—to which all such calculations are liable.

Such a preference appears still more imperatively called for by the results of what are probably deeper, though narrower, inquiries than the scattered and independent records collected and summed up in the foregoing statements. For the larger estimates closely correspond with my own observations in about 25 cases: as well as with those of Lebert[1] in about 35. Indeed, if I might argue from so small a number,[2] my experience would suggest that some of the preceding symptoms occur with even greater frequency. To say nothing of hæmorrhage and cachexia (the frequency of which would scarcely be overstated at 95 per cent.), anorexia, pain, and vomiting are, I believe, all more constant than these estimates suggest. And if it should be objected that the vague and indefinable nature of these symptoms perhaps throws doubt on this suggestion; or that their frequency as elements of ordinary gastric derangement renders it doubtful whether, even when occurring in gastric cancer, they are always due to the lesion; it might be answered that there is still one symptom left, against which all such objections are invalid; and which yet affords the strongest confirmation of this clinical (as opposed to a mere statistical) view. It is precisely in respect to the evidence afforded by a tumour in the belly, that I find the widest divergence between the cases I have seen and those I have collected. In the former, I have rarely failed to detect a tumour long before death; in the latter, it is only by the correction implied above that the 54 per cent. deduced from mere statistics is raised to the proportion of 80 per cent., which is suggested by Lebert's inquiries. In short, I believe that even the large estimate of this able authority understates, rather than overstates, the frequency and the distinctness with which a tumour can be verified by a careful physical examination.

Such being the probable bias of the above estimates, it may be interesting if we attempt to determine their collective bearing on the probabilities of a single case. So much has been said about the obscurity of gastric cancer, that I really think it but right to inculcate the claims of scientific medicine in respect to the diagnosis of this malady: claims which, if thus deducible from these records, will[1] *a fortiori* apply to those strict clinical researches which I hope many of you are preparing yourselves to carry out in after life.

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[1] "Traité Pratique des Maladies Cancéreuses." Paris, 1851.

[2] About a hundred cases since added to my experience confirm this opinion. (Second Edition.)

The selected cases on record afford the following estimate—

	Anorexia.	Pain.	Vomiting.	Hæmorrhage.	Tumour.	Cachexia.
Present .	91	189	193	81	128	3 times,
In . . .	115	205	221	202	159	4 cases.

Applying to these figures the simpler laws of probabilities, brings out the following statements.

The probabilities are eleven to two against all six symptoms being simultaneously present in any given case. In other words, one or other of the six is absent in more than five out of six examples of the malady.

Supposing the least frequent—hæmorrhage—to be absent, the odds are still as many as eight to five against the concurrence of the remaining five.

It is only the absence of a second symptom—for example, cachexia—which equalizes the chances, so as to render exactly even the probabilities for and against the concurrence of the remaining four—pain, vomiting, anorexia, and tumour. In other words, the simultaneous presence of four of the above symptoms seems to be the average amount of evidence detected in the recorded cases of gastric cancer. The numerical frequency of anorexia and cachexia (70 and 75) is so nearly alike, that it scarcely matters which we include in this four; save that, on the whole, the presence or absence of cachexia is less mistakeable than that of anorexia. Hence supposing, in any given instance, we find pain, vomiting, tumour, and cachexia are all present, we may feel that the diagnosis, even if uncertain, is as little so as, in the majority of cases, it is likely to be.

The chances of concurrence would of course increase as the number of symptoms decreased. Thus it is 23 to 12 that pain, vomiting, and tumour are all present; $52\frac{1}{2}$ to 12 that pain is associated with vomiting.

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[1] The more so, indeed, since the nature of some of these cases suggests that they were recorded precisely because their symptoms were obscure and deficient.

A more practical hint is derived from an inquiry into the probabilities of a concurrent failure or absence of symptoms. Thus the odds are 2 to 1 against the simultaneous absence of hæmorrhage and cachexia; 2½ to 1 against the failure of hæmorrhage and anorexia; 40 to 1 against the absence of tumour and vomiting; 60 to 1 against the absence of vomiting and pain; 65 to 1 against the absence of tumour and pain; 225 to 1 against the absence of the more obvious and important symptoms of pain, vomiting, hæmorrhage, and tumour; and lastly, about 2560 to 1 against the absence of all the six symptoms tabulated above.

In alluding to such calculations, however, we must remember how large a variation of probabilities could be produced by a slight alteration in the original estimates. Supposing, for example, that we substituted the lower of the two estimates mentioned for most of the above symptoms, on the assumption that they were absent in every case in which they were not mentioned, the difference would not amount to much for each of these symptoms separately. But its results would of course diverge far more widely; so that, for example, the odds would be about 110 to 2 (instead of 11 to 2) against all six symptoms concurring in any single case. The uncertainty which attaches to all calculations founded on limited numbers may seem still more obvious. But in even as few as 200 cases, this element of contingent error becomes comparatively small. Indeed, the actual collation of these 200 cases singularly confirms the above calculations.

On the whole, I think we may sum up the preceding inquiry by the statement, that the more extensively and closely we consider the symptoms of this malady, the more secure does its diagnosis become. With reasonable care, a physician need rarely be unable to recognise a cancer of the stomach; and will generally find, when its presence is long doubtful, there it is in reality absent.

But while I venture to believe that gastric cancer without symptoms is exceedingly rare, I must add that it is anything but infrequent to find this malady simulated by other diseases. Among these, for obvious reasons, the maladies of the liver, the intestines, and the mesentery, and other neighbouring parts, only yield in closeness of resemblance to those other gastric lesions which, in others of these Lectures, are expressly contrasted with cancer.

To this I may add, that not only must grounds for a definite opinion be sometimes wanting throughout the whole history of a cancer of the stomach, but that a similar obscurity far more frequently veils the disease in the earlier parts of its course. As already pointed out, in the phenomenon of gastric cancer, time is everything. Hence the physician who examines a chronic case, or (what is practically the same thing) sees the malady only in its early stage, must often remain in complete uncertainty respecting its nature, until the progress of the disease, increasing (in something like a geometrical progression) the number and distinctness of its symptoms, enables him to come to a decision. Indeed, it is hardly too much to say, that this peculiar mode of accretion of the symptoms often forms a specific aid to our recognition of the disease.

Time forbids me to discuss the diagnosis of the various complications of gastric cancer. As pathological incidents or events of the malady, they will be mentioned in the next section. Their symptoms are, on the whole, less distinct than those of the ordinary diseases which affect the organs they respectively occupy. Indeed, the late period of the gastric malady during which they usually occur, brings them into a coincidence with a degree of prostration such as obscures these symptoms, alike in respect to their own prominence, and to their reaction on the organism generally.

PATHOLOGY.

In discussing the pathology of cancer of the stomach, I shall follow as closely as possible the method adopted with respect to the pathology of gastric ulcer in the preceding Lecture.

The cancerous disease being far less frequent than the ulcerative, I have had, if possible, greater reasons for adding, to my own personal experience, whatever could be contributed by researches amongst Hospital records, Pathological Museums, Reports, and Journals, respecting it. These inquiries seem to afford a broader (and therefore better) basis for deductions than any previously published.

The frequency of the malady cannot be accurately deduced from mortuary returns; although the statistics of Tanchou in Paris, D'Espine in Geneva, and the Registrar-General in this country would afford a vast array of figures as materials for such a deduction. Unless founded upon necropsies, such returns are useless for pathological inquiries like that we are now engaged in. For though, from the fatal character of the malady, we might with little error regard the number of persons whom it attacks as exactly corresponding with the number of deaths caused by it, yet the vagueness and inexactitude of the opinions which such returns often include and sum up, as well as the errors which are frequently made in the mere nomenclature of disease, invalidate all their conclusions. Indeed, to the general uncertainty of diagnosis, we must add another special and obvious source of error, which would, on the whole, greatly increase the apparent frequency of this particular disease. The frequency and fatality of the gastric ulcer, coupled with the close resemblance its symptoms often exhibit to the cancerous disease of the stomach— a resemblance which it sometimes baffles the most sedulous observation to unmask during life—render it more than probable that, in returns which scarcely attempt to distinguish the numbers of deaths produced by each of these maladies, the mortality attributed to gastric cancer is always far too great. In like manner, that interstitial inflammation of the stomach often improperly termed "hypertrophy" (Lecture V.), may easily be mistaken for cancer; and, though a far less frequent source of error, is still not unworthy of notice.

Hence it is to large numbers of necropsies, the subjects of which have died from all causes indifferently, that we must look for information on this head. From various sources—but especially from the valuable records kindly placed at my disposal in Guy's, St. George's, St. Thomas's, and St. Mary's Hospitals —I have collected a total of 8468 necropsies, which include 81 cases of primary cancer of the stomach; a proportion nearly equivalent to 1 per cent., or $\frac{1}{100}$th of the total mortality.

The proportion borne by cancer of the stomach to cancer of other organs is one which it belongs rather to the pathology of cancer than of the stomach to determine. But from researches by Lebert,[1] Willigk,[2] and myself,[3] it may be concluded that the gastric lesion forms about one third of all primary cancers; and equals (if not exceeds) the number occupying the uterus and breast.

As regards the age of its occurrence, I have collected 600 cases, the age of which at death averages 50 years. The greater part ($\frac{3}{4}$ths or 435) of these 600 cases is claimed by the epoch of life between 40 and 70. Arranged in decades of years, the maximum number ($\frac{2}{7}$ths or 162) occurs between 50 and 60.

Comparing such absolute numbers with the numbers of persons living in these decades of life, we obtain an estimate of the relative liability of the corresponding ages to the malady.

The maximum liability is between 60 and 70. Up to the age of 20, the whole risk is less than $\frac{1}{50}$th of what it reaches between 20 and 30. The latter liability, again, is multiplied in the following decades of years by 3, 6, 8, and 10 respectively. The maximum then seems to sink to little more than half for the two next decades, ending at the extreme age of 100.

It would thus appear that, before the age of 40, the liability scarcely attains one fifth of its total amount: in other words, four fifths of this risk still remain, to be encountered in the succeeding years of life. At the age of 60, nearly one half the risk of the malady has already gone by: and at the age of 70, two thirds. The small number of cases, as well as of persons living, in the last two decades of life, render the conclusions that refer to these epochs somewhat less trustworthy.

Comparing the risk of gastric cancer with that of gastric ulcer,[4] it would seem that the former is on an average barely one fourth of the latter; and that, though much more distinctly and exclusively a disease of old age, its climax or maximum of risk occurs at least twenty years earlier than that of the gastric ulcer. Comparing gastric cancer with apoplexy, it would seem that the risk of the latter disease, beginning about ten years later than that of the former, remains, with little deviation from the proportion of one half the former, until the liability to cancer has reached its climax: when the risk of apoplexy, rising to its own maximum in the next ten years, reverses the above proportion; and at length, between the age of 80 and 90, subsides to a death-rate which is nearly equal for three such diverse diseases as phthisis, cancer, and apoplexy.[1]

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[1] "Traité pratique des Maladies cancéreuses," p. 97. Paris, 1851.

[2] "Sections-Ergebnisse, Prager Vierteljahrschrift," x. 2. 1853.

[3] "British and Foreign Medico-Chirurgical Review," Jan. 1857.

[4] It must be recollected that the ulcerous lesion differs from the cancerous in

Sex.—The influence of sex is far more difficult to estimate than that of age. For any sufficient numerical basis is still wanting. Death-rates are useless, from the reasons already mentioned. The personal experience of private practice is too small: and even if many observers were to group and unite their experience, still its results would be too promiscuous and uncertain. And in public Institutions for the cure of the sick, the numbers, however large, are useless for all purposes of comparison, unless it be clearly stated what is the average proportion of male to female in the patients among whom the cases of gastric cancer have occurred.

Such considerations well explain the contradictory results arrived at by different authorities; and justify our dismissing them with a brief notice.

Of about 250 cases treated in British Hospitals, I find that the male are to the female as 2 to 1. But I believe these institutions admit more males than females (6 to 5 ?) In nearly the same number collected by Dittrich[2] and Willigk,[3] this excess is reversed into a proportion of 3 males to 4 females. But the institutions from which these cases are derived apparently admit females in far greater numbers than males.

Adding together all the trustworthy returns I have been able

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not being always a primary or efficient cause of death. In other words, it is often found in the dead body without our being able to regard it as having exerted more than a partial causative influence on the fatal event. (See pp. 109, 154.)

[1] For details and illustrations of these statements, the reader is referred to the author's Essay already cited. ("British and Foreign Med.-Chir. Rev." Jan. 1857.)

[2] "Prager Vierteljahrschrift," 1848, B. i. s. 1. "Die Krebsige Entartung des Magens"—a brief but excellent essay.

[3] *Op. cit.*

to meet with, affords me 784 cases; out of which 440 are male and 344 female. Such numbers leave little doubt that, whatever the exact proportions of the sexes, the male is the more frequently affected of the two: a fact which it is impossible to avoid connecting with the exclusive amenability of the female to the mammary and uterine localizations of cancerous disease. The above numbers nearly correspond to a proportion of 4 to 3, or 56 to 43 per cent., respectively.

The mode in which age is affected by sex I deduce from 223 cases, which I have collected chiefly from the London Hospitals before alluded to. Of these, 151 are males and 72 females. Distributing them in decades of years, and correcting their absolute numbers by the numbers of persons living at corresponding ages, affords us the following conclusions as to the comparative liability of the two sexes to cancer in the stomach during the various epochs of life.

1. The liability of the male up to the age of sixty maintains a constant excess, which renders it nearly double that of the female.

2. The specific[1] influence attributed to the years which correspond with the close of the fertile or menstrual epoch, is contradicted by the precisely equal rise of risk which the same age brings to the male.

3. The progression in the rise of the risk is the same for both sexes throughout the greater part of life: being treble that of the previous decade (20 to 30) between 30 and 40, double this latter from 40 to 60 years of age.

4. The period of life after the age of 60 introduces a remarkable contrast to the above law. The numbers between the age of 70 and 90 are perhaps too small to justify a safe conclusion. But it seems quite certain that the 10 years between 60 and 70 bring about a converse alteration of risk in the two sexes,—increasing that of the male to double, but diminishing that of the female to half its former amount. Whether this change really represents a corresponding alteration of risk from cancer in general, or whether it is in any degree equalized by an increased liability of the female to the

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[1] Specific, that is, in the sense of being supposed due to regress or involution of the female generative system.

mammary and uterine localizations of the disease, must be left to future researches to determine.

The average age in the two sexes is about 51 in the male, 40½ in the female.

As regards the exact *situation* of the cancerous deposit in the stomach, out of 360 cases collected and compared by me, the pylorus was affected in 219 instances—a proportion of exactly 60 per cent., or three-fifths of the whole. The sex of the patient seems to have no influence on the frequency with which the lesion affects this part. Thus, in 232 British cases, concerning which I obtained fuller details than the others, the sexes are to each other nearly as 2 males to 1 female (159 to 73). And this group of cases yields 194 which specify the exact site of the cancer; and includes 125 lesions of the pylorus, which are divided, in nearly the same proportion, into 86 males and 39 females.

The same group of cases also affords a basis for some specific conclusions which are rendered important by the authority of the eminent pathologist whose opinions they somewhat modify. Rokitansky[1] states that "the cardiac orifice of the stomach is but seldom the seat of cancerous degeneration, and it is a circumstance worthy of note, that the pyloric cancer is exactly bounded by the pyloric ring, and never reaches beyond to the duodenum; while that of the cardia—even when it does not descend from the œsophagus above—always spreads itself over a considerable piece of the œsophagus."

Now, firstly, it is only in a qualified sense that we can accept the above word "seldom." For out of the 360 cases already alluded to, no less than 36 are cancers of the cardia; a proportion amounting to exactly 10 per cent. of the total numbers; and to 16⅔ per cent., or 1 in 6, of the pyloric cancers (216). And in British cases which I have collected, the proportion of cardiac cancers is still larger: namely, 25 in 194, equal to 13 per cent.; or 1 in 7 of the whole number, and 1 in 5 of the pyloric lesions. From these cancers of the cardia, I have excluded all cases except those in which the orifice itself was either solely or chiefly affected. And although it is possible that some of them may have been originally developed around the œsophagus, and only extended to the stomach, yet this objection, which applies equally to all the above cases, can scarcely be regarded as a frequent explanation, in the absence of express evidence to that effect. We must therefore conclude that the cardiac orifice is *not* an infrequent situation of cancer in the stomach.

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[1] "Handbuch der speciellen Pathologischen Anatomie," Bd. ii. s. 205.

Secondly, it is curious to notice how specifically the observations I have brought together contradict the next proposition of the accurate pathologist quoted above. The 125 cancers of the pylorus included no less than 10 cases in which the disease was *not* bounded by the valve, but passed beyond it for a variable distance (often an inch or two) into the duodenum. Lebert[1] gives another instance of the same kind: making in all eleven exceptions to this (presumedly absolute and universal) rule.

Lastly, to the equally absolute statement, that the cardiac cancer *always* involves the œsophagus, I have also found two conclusive exceptions. Allowing for the much smaller number of these cardiac cases, it is not impossible that such exceptions may be also as numerous as the preceding. Indeed the proportions in the cases I have collected exactly correspond to such a conjecture; these exceptions being 11 in 159 pyloric cancers, and 2 in 30 cardiac cancers (or about 1 in 15 for each).

Hence the rules which Rokitansky has the merit[2] of having laid down in these respects are (like many others in this branch of natural science) of general, though not universal import. But their value is not much affected by occasional exceptions. For their significance, far from depending on any mere numerical ratio of fifteen to one, is much more essentially measured by the antagonism of the fact in the two classes of lesion: the limitation, and the spread, of the cancer beyond an analogous boundary or extremity of the organ. Hence it becomes interesting to inquire whether any reason can be assigned for it.

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[1] *Op. cit.*, p. 467.

[2] I cannot think it invidious to call attention to any inaccuracies in a work so widely known and appreciated as the great Text-Book of Pathological Anatomy cited above. Precisely because it demands and repays a thoughtful and minute study, and because every sentence evidently sums up a wide series of accurate observations, we may justifiably apply to it a criticism of unusual severity—a criticism which, even if it weigh every word, will scarcely do more than the author's terse and weighty propositions really deserve.

On the whole, I think the present state of our knowledge justifies some conjecture of this kind. The character of the above laws—general, but not universal—sufficiently hints that they depend, not on any primary and direct cause, such as would necessarily be of universal operancy, but rather on some secondary causation, which indirectly brings about its result. And the first and nearest inquiry is one bearing on the anatomy of the stomach in relation to that of the cancerous deposit. The question naturally suggests itself—"Is there nothing about the structure of the pyloric and cardiac orifices which, by opposing or facilitating the continuity of the cancerous deposit in their tissues, may respectively limit or permit its spread to outlying parts of the alimentary tube?"

The answer to this question appears to afford exactly the kind of explanation suggested. The minute anatomy of gastric cancer conclusively shows (as we shall presently have occasion to mention in detail) that, in most cases, the morbid cell-growth is at first deposited in the sub-mucous areolar tissue; that it thence extends, along those partitions of the same tissue which separate and ensheath the bundles of the organic muscle, into the subjacent muscular coat; that, at a later period, it engages the mucous membrane, by disorganizing which it soon produces symptoms of grave (if not fatal) import; and lastly, that the complete implication of the peritoneal coat is a still later phenomenon, which is therefore generally anticipated by death.

Now it is precisely in the degree and kind of continuity by which these first and chief seats of the cancerous deposit—the sub-mucous and muscular tissues of the stomach—merge into their analogues in the adjacent segments of the digestive canal, that the cardia and pylorus differ most remarkably from each other. The cardia is so organized as always to concede to whatever food may be swallowed a transit in the onward direction—a transit during which its contraction implies a muscular movement that is directly continued (p. 10) from the œsophagus into the stomach. In accordance with these requirements, there is the most perfect continuity between the sub-mucous and muscular coats of the two segments. The latter coat, in leaving the œsophagus, radiates its longitudinal layer on all sides, and with special distinctness along the lesser curvature; whilst its circular or transverse layer has an almost equal continuity with both the circular and oblique layers which together represent it in the stomach.

On the other hand, the pylorus is constructed so as to resist the onward transit of the contents of the stomach by a powerful contraction during the whole period of gastric digestion;—a contraction that appears to be but just overbalanced by every wave of the powerful peristalsis which gradually engages the strong muscular layer of the pyloric region, so as to strain off a little of the more liquid contents of the stomach during each of these undulations. The violent action which thus engages the pylorus from the time the food enters the stomach, requires its complete isolation from the adjacent duodenum. And a careful dissection of the pylorus shows that this isolation is attained, to a considerable degree, even for the sub-mucous areolar tissue, by its small quantity, and by its close and dense attachment at the line of junction of the two mucous membranes. But it is especially in the muscular structure that this isolation is most distinct. The mode of attachment of the duodenum to the stomach may be best expressed by the statement that, instead of being continuous with the pyloric extremity of this organ, it is attached around it, at a short distance from its termination—in short, that the commencement of the bowel receives the stomach (p. 8), much as the vagina receives and embraces the neck of the womb at some distance[1] from its mouth. Hence, not only is the continuity of the muscular walls of the two tubes reduced to a thin linear attachment of the duodenal coat, but this delicate layer comes off from the stomach at such an angle, and at so great a distance from its pyloric extremity, as renders it far more likely that the deposit should engage the massy pyloric valve beyond this attachment, than that it should diverge—through what seems to be chiefly

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[1] I am aware that the illustration I have selected may seem to militate against the very conjecture for which it helps to lay the foundation: by showing an equally discontinuous structure, where, nevertheless, cancer often spreads. But we must recollect that, owing to the less direct influence exercised by these sexual organs on the life of the patient, the cancerous lesion is prolonged to a much later period; to one which, at any rate, might well suffice to convert the ratio of continuity of cancerous deposit seen in the stomach (1 in 15 cases), to that which seems to prevail in the uterus (1 in 3).

a second or outer process of areolar tissue—towards the duodenum. This view is confirmed by many analogous phenomena in the distribution of cancer; by the manner, for example, in which sub-peritoneal cancer almost always involves the continuous membrane that covers two or three organs or segments of the digestive canal; by the converse frequency with which the disease, when primarily affecting a gland, leaps over (as it were) a scanty medium of areolar tissue, to fix on a more congenial organ at some distance from its original seat; and especially by the contrasted rapidity and frequency with which the areolar tissue in front of the uterus, and the serous membrane behind it, become involved and perforated by ordinary uterine cancer.[1]

It is interesting to contrast the other situations of the above 360 cancers of the stomach, with those of as many ulcers of this organ—especially as some authorities seem to think that these two diseases affect the pyloric and cardiac extremities of the stomach in nearly equal proportions.

Such a comparison[2] of the two diseases shows that the remarkable preference of the cancerous lesion for the orifices of the stomach (which together form the site of seventy-one per cent., or nearly three-fourths of the gastric cancers), is not at all shared in by the ulcerous lesion. In the recorded necropsies of the latter, the terms "pylorus" and "cardia" so often refer to the mere neighbourhood of these valves, that a large deduction (probably at least one-half) must be made from even that moderate proportion (sixteen per cent., or less than one-sixth) which their numbers would together imply.

The contrast of the remaining situations does not seem to call for any comment. The absence of all cases of ulcer of the whole stomach, or of the whole of its middle region, is explained by the necessary intervention of death, as soon as the process of ulcerative absorption has destroyed a large proportion of a mucous membrane so essential to life as that of the stomach. It may be added, that the cancers of the whole stomach (thirteen in number) have been carefully distinguished from those cases of interstitial inflammation of the gastric parietes which are sometimes mistaken for scirrhous disease; either by direct evidence derived from their microscopic and anatomical characters, or by indirect evidence equally trustworthy, in the form of the presence of secondary cancer in other organs.

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[1] See Dr. West, "Lectures on the Diseases of Women," vol. i. p. 346; and Dr. A. Farre, article "Uterus," Abnormal Anatomy, p. 700, in the "Cyclopædia of Anatomy."

[2] For the sources, and the tabulated details, of this comparison, see the author's Essay already cited, p. 221.

The *anatomy* of gastric cancer is most conveniently subdivided according to the three chief forms of this deposit which it affects—the scirrhous, medullary, and colloid cancer; to which we may add a fourth, villous cancer of the mucous membrane.

Of these varieties of cancer, the scirrhous is by far the most common. Out of 180 cases which name the species of cancer present, 130 (a proportion equivalent to about 72 per cent., or nearly three-fourths of the whole) belong to this variety. The same group affords 32 instances of medullary cancer, 17 of colloid, 3 of melanotic pigment, and 1 of villous cancer. These numbers correspond to percentages of 18 and $9\frac{1}{3}$ for the medullary and colloid cancer respectively; or, in fractions, to rather more than one-sixth and one-twelfth. The melanosis was in one case diffused generally over the whole body, in the form of small tumours; in the two others, was such a superficial coloration of the gastric mucous membrane covering the cancerous tumour, as would scarcely merit this title, unless substantiated by careful histological examination.[1]

As my limits oblige me to confine myself strictly to gastric cancer, I shall not dwell on the histology of these three forms of the cancerous deposit. The less so, indeed, that they merge into each other by gradations of almost infinite variety. For the scirrhous, medullary, and colloid cancer of the stomach, must be regarded as not only what they are in other parts of the body—manifestations, in a different form, of the same disease; but as constituting, with even greater frequency, mere consecutive phenomena of the same local process. For it is evident that, in many cases, a growth originally scirrhous undergoes an admixture of medullary or colloid cancer, if not an absolute metamorphosis into it. While collateral circumstances entitle us to conjecture that in many more a similar complication would occur, but for the occurrence of death in an earlier stage of the disease. Whatever the apparent temerity of such a view, its truth becomes sufficiently obvious when we consider the propositions on which it is based. Our knowledge of the pathological anatomy of this (or any other) disease is built up from a number of observations. Of these observations, many of the most valuable in respect to the origin and succession of morbid changes in the organ forming the seat of the disease are only afforded us, either by casual deaths; by intercurrent or secondary maladies; or finally, by the disease acting on the powers of life with an effect which is so complex and variable, that we may fairly view it as somewhat independent of the local mischief. Other observations, though relating to appearances by no means frequent, must be regarded as showing the more direct and typical effects of the disease; and as therefore possessing an importance far beyond what their numerical rarity, and the late period of their access, would suggest.

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[1] In order to prove that it was not due to any of those alterations in colour which mere ecchymoses in this situation often undergo from the action of the digestive fluids, compare pp. 61, 76.

In almost all cases, the cancer begins as a deposit in the sub-mucous areolar tissue. This loose, but thick layer—which, by intervening between the mucous and muscular coats, shares in every movement of the two; and especially concedes and limits that free play of the mucous membrane which permits the passive contraction of the muscular coat to throw it into folds (p. 14) such as are effaced by any distension of the stomach—is composed of its ordinary constituents of white and yellow fibrous tissues, the elements of the latter being both large and numerous. Among these fibrous structures the first rudiments of a cancer are generally deposited in the form of a dense knot, which has a dull white colour, and a firm and hard (rather than tough) texture. This opaque mass encloses the above normal elements of the areolar tissue, in such an intimate state of fusion, that it is almost impossible to detect them in any quantity. Indeed, their proportion is so small that even allowing for the mechanical difficulties that oppose their isolation, it is difficult to avoid the conjecture that they are compressed and killed by the new growth; in which, so to speak, they remain dead and buried. And it is to the strictly retrograde changes of this kind which these healthy original structures undergo, that I am disposed to attribute the fatty molecules which are found in even the freshest specimens of scirrhus, and in the earliest stages of its growth. In many cases at least, I believe this fatty matter is due to a mere decay of the original tissues. Blastema in any quantity is rarely met with, save in the whitish juice which can be obtained by compression of the scirrhous mass. The irregularly granular character of the layer into which these knots soon expand, seems to be often quite unconnected with any peculiarities in the arrangement of the scirrhous growth with respect to those plexuses of vessels which occupy the sub-mucous areolar tissue (p. 22), though occasionally the denser grains of the mass seem to be received into vascular loops or meshes of this kind. Finally, in most instances, however small or recent the mass of scirrhus, a large proportion of its substance consists either of fibres, or of fusiform cells, the extremities of which are so long as almost to deserve this title. Mere nuclei are in but small proportion.

It is often difficult to determine by examination whether the scirrhous deposit has originally predominated on the muscular or external aspect of this layer of sub-mucous areolar tissue. But there are good grounds for supposing that this is generally the case. At any rate, it is in this direction, and towards this constituent of the stomach, that its progress in the succeeding stage of the disease is almost exclusively directed. The areolar and muscular coats become inextricably fused into each other at their line of contact, while as yet the mucous membrane itself is still separated from the deposit by an interval of healthy areolar tissue. The muscular coat is then transformed into scirrhus by an extension of the same process as that by which the sub-mucous tissue was itself at first affected.

The appearances of the scirrhus in this stage are too specifically connected with the organ it affects, to be left unnoticed. In rare instances, we may find a dense white semi-translucent mass, of tolerably uniform structure, occupying the whole thickness of what was formerly the muscular coat, and quite undistinguishable from the original focus of the disease in the sub-mucous areolar tissue.

But in the majority of cases, the cancerous deposit is much less homogeneous; and exhibits characters which may be regarded as relics of that differentiation of tissue proper to the healthy textures which it has replaced. On cutting through the diseased mass, we find that the deeper portion, which corresponds to the original muscular substance, exhibits two varieties of tissue, so arranged as to give it more or less of a honeycombed appearance on section. There is a pulpy, brownish, reddish-yellow (or at any rate somewhat darker and softer) mass, enclosed in small polygonal meshes formed of whiter and denser tissue. Of these meshes, which are irregular in shape and size, those are generally strongest and thickest which run transversely to the axis of the stomach from the sub-mucous to the sub-serous areolar tissue. They are evidently the transformed septa of those bundles of fibre cells which form the thick muscular coat of this part of the stomach; and they enclose cavities which, originally occupied by these muscular bundles, now contain, in addition to their more or less fatty and decayed relics, a cancerous deposit, often visibly less fibrous, and richer in nuclei and blastema than that of the septa themselves. Their arrangement, however, is not always precisely like that of the original sheaths of the unstriped fibres. The thickest septa are, as above mentioned, often directed transversely to the mucous surface; and from the direction of the circular fibres, are best seen by cutting lengthwise through the mass in the same plane. But irregularities in the deposit of the cancerous matter, as well in the septa as in their enclosed cavities, easily affect the uniformity of this arrangement: so as to render the meshes comparatively larger or smaller, or even to obscure and obliterate them altogether. And as a tolerably equable deposit around the narrow pyloric end of the stomach is one of the most common arrangements of scirrhus in this organ, the first effect of the commencing disease often presents an obstacle to further growth in the direction of the centre of the tube, such as even aids the natural tendency of the disease to spread outwards.

Hence the septa often seem to diverge (or even radiate) in passing from the centre to the periphery of the tumour; and enclose an increasing amount of their darkish or gelatinous-looking contents, as they approach the tissues of the peritoneal coat of the stomach..

There is one circumstance which renders it important to appreciate these characteristic appearances of the gastric scirrhus in this stage of its growth. It is probably not uncommon for specimens which present little more than the average distinctness of the above differentiation of ordinary scirrhus, to receive the name of "colloid" cancer. And though it is true that in many instances of this kind a careful histological inquiry could distinguish the tissue as really fibrous or scirrhous cancer—a decision in which its physical as well as microscopic characters would alike concur—still in some cases, in which the meshes are large and enclose much fluid exsudation, it is not easy to say to which of the two species the cancer really belongs. To some examples, indeed, it would be difficult to deny the appellation of colloid. And, on the whole, I think that we may fairly assert the following propositions respecting what is called colloid cancer of the stomach. (1) That in some cases the colloid arrangement imitates (and indeed perpetuates) the natural differentiation of tissue above alluded to. (2) That in many more the deposit is either essentially scirrhous, or is developed out of a cancerous deposit which was originally of this species. Hence (3) that the ordinary way in which this name is at present used, obliges us to deduct from the recorded cases of colloid cancer of the stomach a very considerable proportion as modified scirrhus. And (4)—what is even a more practical point for the pathologist—that such cases have little or no weight in the decision of an important question in the natural history of gastric cancer; namely, the average duration of the three forms of the malady, or the rapidity with which they severally destroy life.

The further progress of the disease conducts it to the mucous and serous surfaces of the stomach; still, as it goes, fusing into itself, and each other, the structures previously present.

In the serous membrane, the tenuity of its tissues, as well as its pathological tendency to inflammation, soon give rise to adhesion of the cancerous segment of stomach to some neighbouring viscus. How far the cancer itself lends the aid of any specific irritation to this process it is difficult to estimate; although, from the analogy afforded by gastric ulcer, one may suspect that there is no great need of assuming any such explanation. Of course the particular portion of the tumour thus attached, as well as the viscus to which it is fixed, are subject to considerable variety. But while (for obvious reasons) the adhesion of the ulcerous stomach accurately corresponds to the site of the lesion, and to the viscus which is normally in contact therewith, that of the cancerous organ appears to be regulated by circumstances less obvious. The diaphragm, liver, pancreas, and spleen, constitute the most frequent sites of such adhesion; and in nearly the above order of decreasing frequency. In short, it is at the upper and posterior aspect of the stomach, and with a frequency which is quite disproportionate to the predominance of the cancer itself in these situations, that this fixation chiefly occurs. It is probable that this peculiarity has its chief cause in the relative immobility of the organs which occupy this part of the cavity of the belly; and that it is so far analogous (if not strictly parallel) to the infrequency or tenuity of adhesions between an ulcer of the anterior surface of the stomach and the corresponding wall of the belly. Indeed it may fairly be said that the same contrast, which we can only verify in a number of cases of ulcer, is often illustrated by opposite surfaces of one and the same cancerous tumour.

In advancing towards the mucous membrane, the cancer inaugurates a series of changes which, though of secondary nature, and of subsequent occurrence to its own deposit, are probably more intimately connected with those symptoms which generally attract the notice, and claim the skill, of the physician, than any other of the details which the morbid anatomist has to study. The destruction of the gastric mucous membrane not only directly involves the partial removal of an organ essential to life, but leads to a train of indirect results of at least equally serious import. The certainty of hæmorrhage and ulceration; the probability of a grafting of cancerous germs into distant organs by means of the currents of the lymph and the blood: together with an increased possibility of obstruction, dilatation, and hypertrophy of the stomach; as well as of fistula opening into other parts of the canal, the chest, or the surface of the belly:—all these risks are now superadded to those of the cachexia which the cancer itself expresses, and to those of the cachexia which it can produce.

As the cancer approaches the immediate proximity of the mucous membrane, its first effect often appears to be a slight (though perceptible) increase in the thickness and firmness of this membrane, such as a mere excess of its healthy nutritional fluid could probably produce. It resembles, in short, a stomach taken from a younger and healthier person, or from a fresher corpse, than that to which it belongs.[1] Then follows a fusion of the cancer with the under surface of the mucous membrane; giving to this latter tunic a complete immobility upon the subjacent textures; and a dull white, thickened appearance. The local anæmia which this state seems to imply, is probably often due to a complete occlusion or compression of the blood-vessels of the membrane: a state which, by the pressure it throws on the obstructed current of the blood, appears to give rise, occasionally, to a hæmorrhage strictly analogous to that which so often causes death in cirrhosis; rarely, to a varicose state of the veins more immediately concerned. And it is probable that this interference[2] with the vessels (quite as much as any specific tendency of the cancer itself) is answerable for the destructive changes of the mucous membrane which next ensue. This destruction—whether effected by an intermediate stage of dark pulpy or ashy grey softening, such as gradually merges into ulceration; or by the more rapid death of the tissues, piecemeal, in a process of sloughing or gangrene;—in any case, speedily leads to the same result, to the production of a solution of continuity which is bounded exclusively by the exposed or denuded cancerous growth.

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[1] And may be compared in so far to the stomach of diabetes; which, as I described many years ago, from a specimen I examined for the late Dr. Todd, is best summed up as remarkably healthy, and unusually resistant of cadaveric changes. Subsequently, I believe, Dr. Todd and myself were both misquoted as having stated that the tubes were in a state of abnormal distension.

[2] The above paragraph is now curiously confirmed from other sources (compare pp. 163, 202), *Second Edition.*

The subsequent phenomena still permit of considerable variety in different specimens. On the whole, the most frequent change is that in which the periphery of the cancerous mass (and especially that part of it which is denuded by the removal of the mucous membrane) becomes the seat of an infiltration of medullary or areolar cancer. This new deposit generally protrudes into the cavity of the stomach as a spongy mass of variable size and shape, which constitutes what is (κατ' ἐξοχήν) *fungus hæmatodes*,—a soft pulpy fungus, which sometimes breaks down by a process, half sloughing, half suppuration; but oftener associates, with a similar process, such an amount of hæmorrhage as materially to disguise and conceal it. Hence, save to a strict scrutiny or a careful microscopic examination, there is often no perceptible difference between the "coffee-grounds" fluid which in such cases is vomited during life, and that which is expelled from a merely ulcerous stomach. In like manner, the surface and edges of the cancerous fungus itself are sometimes occupied by coagulated and altered blood; which, as such, bears no testimony whatever to the character of the lesion producing its extravasation.

This secondary deposit of medullary cancer seems to be precisely analogous to that secondary form of colloid already mentioned; than which it is, on the whole, much more frequent, and to which it not unfrequently serves as a basis. Both of them suggest the question as to how far they are real transformations of the original scirrhus, how far they are merely admixed with it. But the morbid anatomy of a large number of cancerous stomachs suggests a somewhat different answer for each. Using the term "colloid" in its ordinary sense, we can scarcely exclude from this category cases in which colloid seems to have been developed, by something very like a genuine transformation, out of both scirrhous and medullary cancer. In the former of these two species we have seen that it occasionally (though rarely) perpetuates a normal differentiation of tissue; that it is the morbid analogue, so to speak, of the

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[1] It may be objected, that definite instances of this kind are too rare to justify the confusion of terms that would arise from calling them colloid. The validity of such an objection I quite admit. But I think it would be impossible for any one to analyse and study the more modern records of this disease, without coming to the conclusion that (rightly or wrongly) the name "colloid" cancer of the stomach

sheaths and bundles of the unstriped muscle.[1] In the latter it amounts to the introduction, into a comparatively homogeneous cell-growth, of an exactly similar differentiation; describable in most cases as a formation of delicate balks or partitions of fibres, which are arranged so as to enclose irregular cavities containing a softer mass of cells or rudimentary fibres. On the whole, the ordinary situation of the deposit, whether medullary or colloid, seems to show that it is rarely or never produced by any genuine transformation of the pre-existent scirrhus; and that, though it is not at all improbable that a moderate interstitial deposit of new cancer often admixes a certain quantity of medullary or colloid with even the deepest parts of such a scirrhous tumour, no real conversion of the previous scirrhus ever takes place. At any rate, we have no evidence of such a process: and all the softening that occurs in the scirrhus itself seems explained by the degeneration of those healthy tissues which this adventitious deposit has enclosed within its mass, and by the true cancerous softening to which it is liable, in common with all other forms of cancerous growth.

With respect to the origin and growth of the two other varieties of gastric cancer, in their uncombined form, it need only be said, that the appearances of a large number of specimens suggest that, as a rule, the medullary deposit begins more immediately beneath the mucous membrane, and the colloid beneath the serous membrane, than does the ordinary scirrhus or its combinations. But the exceptions to this rule are numerous.

The villous cancer of the stomach seems to commence by a deposit in the immediate neighbourhood of the basement membrane; and to be—not merely (as its form suggests) an isolated medullary deposit in the sub-mucous areolar tissue, growing by continual accretion on its mucous side, and thus sometimes enlarging to a tumour here, of which the original basis remains a mere peduncle or stalk—but rather a cancer of the mucous membrane itself, the close proximity of which is the chief cause

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is often applied to a very moderate exaggeration of an appearance which few cases of scirrhus of this organ are altogether devoid of. Hence I offer the above remarks chiefly as a clue to these records, and as a hint with respect to the physiology of cancer; though I should be happy if they also called attention to a vagueness (if not inaccuracy) in the nomenclature of this disease.

of its great change of form being associated with so little direct injury to its structure. That, in the majority of instances, the deposit lies beneath the basement membrane, the microscope leaves no reason to doubt; a proposition which at once establishes an important distinction between it and the epithelial cancer which has been sometimes regarded as its analogue.

It is only by contrast with such remarks that we can accept or interpret the preceding statements as to the numerical frequency of the three forms of cancer in the stomach. In point of fact, these numbers probably represent little more than instances in which the corresponding variety of cancerous deposit formed a large or predominant portion of the whole mass. Of the 32 instances of medullary cancer, for example, it is probable that several were mere combinations or admixtures of this growth with what was originally and essentially a scirrhus. And of the 17 cases of colloid, perhaps even a larger proportion ought to be referred to a similar category. This conjecture is confirmed by the observations of Dittrich,[1] who found only 3 out of 11 cases of colloid to be pure and uncombined examples of this variety of cancer; the remaining 8 being combined, 7 with scirrhus (2 of these with medullary cancer also), and 1 with medullary cancer.

The cases I have selected afford 34 examples of colloid in 417 of gastric cancer. If the proportion observed by Dittrich were applied to these 34, it would reduce them to 9 cases of pure alveolar cancer; a change equivalent to a reduction from $8\frac{1}{6}$th to $2\frac{1}{6}$th per cent.

The destructive process which ultimately engages the free or internal aspect of a gastric cancer, after its mucous membrane has been removed by the processes already mentioned, is of especial interest, from its close connexion with the appearances by which the cancerous and ulcerous stomach sometimes resemble each other.

Perhaps the most frequent way in which cancer simulates ulcer of the stomach, is that brought about in the course of the process described by Rokitansky; in which (generally with little or no previous formation of medullary or areolar cancer) the scirrhus, denuded by ulceration or sloughing of the mucous membrane, gradually sloughs in round circumscribed patches, or exfoliates in successive strata, so as to excavate a tolerably smooth pit or fossa in its mass. Here, however, the distinction is rarely difficult. The bottom of the pit remains more or less sloughy; or even if this characteristic appearance is disguised by a casual hæmorrhage, such as might easily complicate an ulcer, still the quantity of the surrounding mass, and especially its histological characters as seen under the microscope, will rarely allow of any doubt. Besides, in the ordinary ulcer, thickening is scarcely compatible with sloughing; and is almost always associated with a history which sufficiently refutes the notion of malignant disease.

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[1] *Op. cit.*, p. 22.

But while, in the majority of cases, a careful examination scarcely allows any hesitation as to the ulcerous or cancerous nature of the lesion, there are rare instances in which even the most sedulous examination of the dead body reveals nothing to justify a decision. Thus a smooth and circular excavation in a scirrhus may obviously imitate an ulcer surrounded by a hard and thickened mucous membrane. And if the cancerous hollow be for the time denuded of slough, or covered with blood, we lose another of the means of distinction which a mere inspection can afford us. Nor will even the physical and microscopical characters of the hardened periphery of the sore always decide the question. In other words, though in my own experience the total necropsy has never yet left me in doubt, still the dense cicatrix round an ulcer has sometimes yielded portions respecting which, had I seen them separately, and been obliged to decide their character solely from their microscopic and other appearances, I could scarcely have come to a decision. In respect to the microscope, indeed, the great variations in the quantity of cell-growth contained in a scirrhous tumour, and in the developmental stage attained by its fibres, will sometimes render its diagnosis from the more complete forms of cicatrix-tissue (containing a fair proportion of fibres and long fusiform cells) anything but an easy task.

Such equivocal cases present themselves in a still more marked form in some of the numerous records and specimens to which my researches have led me. This form, however, ought rather to be suggested as a contingency, than announced as a fact. On the whole, it seems possible that the sloughing or ulceration of a small and circumscribed deposit of scirrhus may not only imitate a mere ulcer, but may produce or become one;—and that, in rare instances, the destructive process may remove the whole of the malignant deposit present, leaving behind it what appears to be neither more nor less than a circular ulcer, bounded by healthy (or at most inflamed) tissue.

Such a contingency is especially suggested by one or two instances, in which a lesion offering every appearance of a simple ulcer, with a but slightly thickened (or even healthy) margin of mucous membrane, has been associated with deposits in the liver and lungs, which (if not absolutely cancerous) at any rate closely simulated secondary cancer. But having hitherto had no opportunity of a minute examination of these ulcers and deposits, I am obliged to leave the question in its present form,—as a mere suspicion, calling for further inquiry.

As regards mere combinations of cancer and ulcer, little need be said. Since the gastric ulcer has no protective influence against cancer, we need scarcely wonder to find that its scars are often found in stomachs which have subsequently been attacked by the deadlier malady. The coincidence of the open ulcer with cancer is scarcely less frequent. But, as might be expected, in these cases also it is the cancer which is added to the ulcer; and never *vice versâ*—the ulcer to the cancer. Indeed, the formation of an ulcer in a part of the cancerous stomach unaffected by this growth, is, so far as I am aware, quite unknown in the history of gastric cancer. Nor are there any authentic instances in my knowledge, where an ulcerous stomach has been attacked with malignant disease, without the ulcer itself becoming implicated, to at least such an extent as to have its base or edges infiltrated with the new deposit. More frequently, indeed, it is these parts alone which become the site of the cancerous deposit. An ulcer, for example, which has lasted many years, suddenly ends in death;—the necropsy revealing a considerable cancerous infiltration in the thickened margin of the ulcer, or in that more chronic thickening of the walls of the stomach which often extends some distance beyond this margin; or showing a fungous mass, of comparatively recent growth, springing up from the centre of the excavation. In rare instances a similar marginal infiltration of cancer seems to have occurred after the original ulcer has perforated the stomach; thus involving the parietes of that chronic abscess to which, under favourable circumstances, perforation (p. 184) gives rise. And while there seems to be scarcely any limit to the varieties of this kind which may occasionally occur, still, on the whole, the relative frequency of such combinations is quite sufficient to suggest that the presence of an open ulcer sometimes provokes the development of the cancerous cachexia; as well as aids in determining the deposit of cancer in this particular organ.

We shall hereafter (Lecture V.) compare with the gastric cancer another disease which, though not known to combine with it, often simulates its appearances to such a degree as to be mistaken for it. And though such an error is practically of far less importance than that of confounding cancer and ulcer—because, unlike the latter of these two diseases, the peculiar inflammatory thickening of the stomach to which we are now alluding seems scarcely less fatal, or more amenable to treatment, than cancer itself—yet the pathological distinctness of the two lesions seems to be just as complete, and the typical course of their symptoms just as diverse, as is the case with cancer and ulcer.

The chief characters which distinguish this lesion from scirrhus may be enumerated as follows. In marked specimens, the change involves the whole of the stomach in a moderate thickening which, while it allows the three coats to be still distinguished and separated from each other, increases their bulk in a tolerably equal proportion. The uniform expanse of stomach involved in the change is yellow, tough, and elastic: instead of having the peculiar pearly-white appearance, and gristly section, of cancer. The muscular tissue is almost always discernible: and is generally hypertrophied in the earlier stages of the disease. And not only does the perfectly homogeneous deposit offer none of those varieties which commonly mark the later progress of a cancerous deposit (in the shape of colloid and medullary matter surrounding the original and central scirrhus), but its microscopic examination shows either an absence of the characteristic cells of cancer, or a few spindle-shaped cells of fibrous import, scattered sparingly throughout a mass of laminated (but scarcely otherwise organized) exsudation. The absence of secondary cancer in other organs aids the diagnosis of this benignant lesion. Ulceration, too, is less early, frequent, and extensive, than in cancer. Finally, the pathological contrast of the two diseases is rendered complete by cases which suggest that the lymph thus deposited by a kind of cirrhotic inflammation around the gastric vessels occasionally undergoes a development into cartilage; rarely even into bone.

The presence of *secondary cancerous deposits* in other organs is a very frequent complication of cancer of the stomach. Out of 437 cases, 210 (equivalent to 48 per cent. or nearly one-half) exhibit this complication.

As regards the comparative liability of the different varieties of cancer to be accompanied by secondary deposits, it would seem that the scirrhous, medullary, and colloid are associated with secondary cancer, in proportions nearly equivalent to the respective fractions of three-sevenths, four-sevenths, and six-sevenths. In other words, nearly one-half the cases of gastric scirrhus are associated with a deposit of secondary cancer in some other organ of the body; and this proportion, which is increased by one-third in the case of medullary cancer, is doubled in that of colloid. This fact constitutes at least a partial explanation of the greater and more rapid fatality of the two latter forms of gastric cancer.

The cancer which forms the secondary deposit is generally of the medullary variety. But in the liver or peritoneum, it sometimes affects the areolar arrangement of colloid. A similar arrangement, which is sometimes met with in the lymphatic glands of the belly, appears due to a differentiation akin to that of the original structures.

As regards the influence of sex on these secondary deposits, it would seem that they are about one and a half times more frequent in the male than in the female subjects of gastric cancer.

Among the organs occupied by these secondary deposits, the liver claims that precedence which might be expected. Out of 431 cases, 105 (about 25 per cent., or one-fourth) exhibited a cancerous deposit in the liver: a proportion at least twice as great as that of such deposit in the lymphatic glands adjoining the stomach, and thrice as great as that of secondary cancer of the lungs.

In many of these cases, the presence of cancer in the liver was associated with its deposit in other organs. The only exact numbers I have been able to obtain in respect to this further complication, are derived from 47 instances of secondary hepatic cancer, occurring in 214 cases of cancer of the stomach. Out of these 47 instances, such a coincidence was present in 13: nine times in the glands or peritoneum adjacent to the stomach, twice in the kidney, and once in each of the following organs—the ovary, pancreas, intestine, spleen, thoracic glands, and lung. The close proximity of the abdominal glands and peritoneum renders it difficult to lay much stress on their frequent share in the hepatic mischief. But of the remaining situations of the deposit, it is interesting to notice that the lung is affected with only one-fourth or one-fifth of its average frequency: while the abdominal viscera are involved about one and a half times more frequently than their average stated below. In other words, it seems as though the secondary deposit of cancer in the liver somewhat increased[1] the chance of other abdominal viscera sharing in the deposit; while it much more decidedly diminished the risk of pulmonary ingrafting of the disease.

It is sometimes so difficult accurately to distinguish secondary deposits in the lymphatic glands which adjoin the stomach, from similar deposits in and between the layers of peritoneum which form the gastro-hepatic and greater omenta, that it has seemed best to group them together. Adopting this arrangement shows these structures to be involved in about $25\frac{1}{2}$ per cent. (or rather more than one-fourth) of 271 cases of gastric cancer.

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[1] Of course it is possible that, in some of these cases, the hepatic deposit is secondary to the visceral: being produced, for example, by the reception of cancer-germs into the portal system from a mesenteric deposit. But without implying any real causation, it seems best, for diagnostic reasons, to give the hepatic deposit the precedence.

The lungs seem to be affected about 1 in 12 times, or in $8\frac{1}{6}$th per cent. of the total number of cases. This estimate is based on 35 and 431 cases respectively.

But in adducing these numbers, it becomes imperative to consider some circumstances which materially detract from their value; the more materially that, in doing so, they furnish no data for any correction of the errors they indicate.

And firstly, the cases to which these numbers correspond omit all mention of a pulmonary lesion which, in the shape of pleurisy, pneumonia, or both these diseases in combination, occurs so frequently in connexion with cancer of the stomach, that it is impossible to doubt its general significance in relation to the primary disease. The lungs exhibit, in some part of their mass—usually in the lower lobes, and more frequently (I think) in the left than in the right organ—a kind of reddish-grey hepatization; which generally extends to the nearest pleural surface, and gives rise to more or less adhesion here. The adherent tissues are united by a small quantity of lymph, of very moderate tenacity, and pasty (rather than fibrous) consistence. And the pleural cavity itself is often occupied by a variable amount of serum; much of which, however, is doubtless in some cases effused during or after death: of which event this pleuro-pneumonic complication seems to be no very unusual cause.

Another (and still more frequent) complication of gastric cancer, relates to the association of what is called "tubercle" with the primary disease;—an association of such remarkable frequency as to raise suspicions akin to those suggested by the above pleuro-pneumonic complication.

It is the more important to institute some inquiry into the facts which suggest these suspicions, inasmuch as they are intimately connected with the general pathology of the cancerous and tubercular deposits. And even limiting this inquiry to the malady we are now discussing, we may, I think, point out some errors in the facts on which are built the existing doctrines relative to these two comprehensive forms of disease.

The incongruous ideas prevalent with respect to these two diseases are well illustrated by the statement that, while Rokitansky has long regarded them as almost exclusive of (or incompatible with) each other, so large a number of cancerous necropsies reveal tubercles in the lungs or other parts of the body, as almost to confirm, relatively to individuals, the propositions stated by Dr. Christison[1] relatively to families: namely —that "the malignant diseases belong to the scrofulous constitution;" and that "consumption in early life, and malignant disease at a later age, seem not infrequent in the same family." In short, it would seem that not only do the two diseases attack similar constitutions and temperaments, but that they often merge into each other in the same individual; and that, less frequently, their respective products are present in a quantity and condition which conclusively indicate a simultaneous—if not indeed a connected—activity of both maladies.

But in respect to the mutual relation of cancer and tubercle, the appearances of the malady we are now discussing seem, if carefully considered, to support Rokitansky's views.

Firstly, as to the mere presence of the products of cancer and tubercle in the same person. The comparative fatality of the two diseases, and the different epochs of life which they specially affect, are circumstances which, apart from all others, sufficiently explain why cancer follows tubercle, but tubercle does not follow cancer. And the frequency with which arrested or obsolete deposits of tubercle are found scattered in sparing quantity throughout the lungs of persons dying of cancer, is a fact which not only fails to establish any essential connexion between them, but is quite compatible with Rokitansky's views. Whatever the estimates of different observers as to the frequency of tubercular deposits in necropsies of persons dead from all causes indifferently, there can be no doubt that their average (50 per cent.) far exceeds the frequency of similar deposits in cancer. Hence all that we are really entitled to infer is, that the deposit of tubercle in the earlier epochs of life affords no complete protection against that of cancer at a subsequent period: a proposition which few would question.

Of course it might be argued that the blood-disease, by which the tubercular deposit is produced, may sometimes survive the activity of this deposit itself: and hence that of these cases of coincident deposit, some are ascribable to a concurrence of the cancerous and tuberculous cachexiæ.

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[1] "Suggestions to Medical Referees of Standard Life Assurance Company," pp. 11, 12. Edinburgh, 1852.

It is difficult to refute vague suggestions of this kind. But the histology of gastric cancer and its complications throws great doubt on them: and certainly suggests, that a large proportion of what are supposed to be tubercular deposits associated with cancer of the stomach, are in reality cancerous deposits, having the usual secondary relation to the gastric lesion. Thus there are some cases of secondary cancer of the lungs which closely simulate ordinary tubercle, not only in the earlier stages of its deposit, but even at the commencement of its suppuration and softening. A large proportion of the secondary cancerous deposits which involve the lung, possess, either a firm and cartilaginous texture akin to that of ordinary scirrhus, or a somewhat less dense (but still tolerably firm), white, solid appearance, such as can scarcely be mistaken for either miliary or crude tubercle. But in less frequent cases, the cancerous deposit, sometimes equally discrete and scanty, undergoes a process of softening and suppuration which is hardly distinguishable, in certain of its small spherical granules, from the appearances of the similar change in tubercle. Nor does even the microscope always afford a decision. As a rule, the medullary mass of which such secondary cancerous deposits are composed may be easily shown to consist almost exclusively of cytoblasts or minute cells, which occasionally distend the pulmonary lobules so as to afford a complete demonstration of their arrangement. While in tubercle we find, in addition to what are often characteristic differences in the cells themselves, such a preponderance of the amorphous constituent, as is, on the whole, even more distinctive of the nature of the mass. But occasionally these grounds of distinction altogether fail us. The process of softening breaks down the structure of the cancerous deposit, to a degree which sometimes leaves scarcely more of the malignant cell-growth that formerly distended the pulmonary lobules, than might be readily mistaken for the epithelium plentifully found in recent crude tubercle. And the cancerous deposit so closely imitates the structure of the normal epithelium of the pulmonary lobules, that the individual cells, if seen apart from each other, would perplex the most experienced histologist. In short, there are cases in which it must be confessed that, though the microscope suggests a deposit, which at first sight closely resembles tubercle, to be really cancer: still it only decides the question with the aid of the symptoms and history of the case, and the appearances detected in other parts.[1]

In like manner it may be noticed, that Rokitansky[2] specifies a variety of "croupy tubercle of the lung, which occurs during the inflammation and suppuration of cancer, as a result of the cancerous degeneration of the fibrin; and which is distinguished by its whitish colour, its soft glutinous consistence, and by its breaking up into a creamy, whitish ichor." But while I can confirm this statement, I would add, that whatever the predominant appearances of this mass may sometimes be, it is not a croupy tubercle; but, on the contrary, is neither more nor less than a pulmonary deposit of secondary cancer, and as such, consists originally of cell-growth. For in favourable specimens, the various masses of this cell-growth may be seen offering all the degrees of such a croupy character in the same lung: the smaller nodules being white, dense, or even semi-cartilaginous; the larger, either softened in their centre, or completely broken down, and even partially emptied through a neighbouring bronchus. And instances are not wanting which seem to indicate that—though it is easy to imagine that the free access of air to such pulmonary deposits constitutes a chief cause of the proneness to suppuration of this form of secondary cancer, as contrasted with similar deposits in less exposed organs—still the more immediate and effective impulse to the process of softening is given by the perishing of the original lung-tissue, which is cut off from the sources of its nutrition by the mass of adventitious cells that surrounds and encloses it.[3]

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[1] From my own clinical researches, I am quite entitled to affirm, that these discrete and scanty deposits of secondary cancer in the lungs sometimes not only soften and suppurate, but complete their resemblance to tubercle by being actually expectorated, so as to form small cavities. And it would not be difficult to explain the rarity of this event, from its requiring a concurrence of some unusual circumstances, both of the primary and secondary lesion. (Compare "Transactions of the Pathological Society," vol. vii. p. 70.)

[2] *Op. cit.*, p. 70.

[3] In this comparison of tubercle and secondary cancer of the lung, I have pur-

Whatever of novelty or truth this view possesses, it is not for me to decide. But I believe that careful inquiry will in great measure confirm it. In any case, I think that a thoughtful study of the pulmonary appearances of a series of cases of gastric cancer brings us to this alternative:—either that the two diseases really have much of that exclusive relation to each other which Rokitansky deduces; or (what is at least as startling a conclusion) that they have an intimate causal relation, such as has never been suspected. The frequency of their apparent coincidence demands an explanation of one kind or the other.

It can scarcely be doubted that these pulmonary deposits of secondary cancer are liable to another change: namely, to an obsolescence; which dries up their mass of cell-growth, and conducts it, through various grades of solidification, to what is, in rare instances, a cretified substance closely resembling that so frequently found as a relic of tubercle in the lung. But of the frequency of this process, as well as of the pleuro-pneumonia and softened cancer just mentioned, I can offer no numerical estimate. And in dismissing this part of the subject, let me say, that the caution I have suggested with respect to the histological evidence sometimes furnished by the microscope, is not in any degree intended to depreciate the value of this indispensable means of research; but rather to guard against the errors into which a hasty and illogical use of that evidence, or an exclusive attention to it, would occasionally lead us. If ever the disease now regarded as a single one under the name of pulmonary tubercle, should be distinguished into several different maladies—a contingency which seems by no means improbable—it is difficult to avoid the suspicion that clinical research will be at least as instrumental in this result as mere morbid anatomy.

The other organs which form the seat of secondary deposit in gastric cancer, scarcely deserve enumeration. The intestine was thus affected in 7 out of 431 cases; twice in the small intestine, twice in the colon, and thrice in the rectum. The same cases also afford six instances of deposit in the ovary;

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posely avoided the term "infarction," much as the earlier appearances of both deposits suggest its use.

three in the uterus, spleen, and pancreas; two in the kidney, the bladder, and the ribs; and finally, one in each of the following organs—the vertebræ (fourth lumbar), the sternum, the humerus, the supra-renal capsule, the thoracic duct, the seminal vesicle, the diaphragm,[1] and the pericardium. Among the rarer secondary deposits is the obstruction of the vena portæ by a soft cancerous mass. This condition, which was present in three or four of a similar deposit in the liver, for obvious reasons, is accompanied by much ascites.

The remaining pathological phenomena of gastric cancer may be next briefly adverted to, in the usual order of their occurrence.

The *obstruction* produced by a cancerous thickening of the gastric parietes often gives rise to a variable degree of one or more of the following conditions:—(1), hypertrophy of the muscular coat; (2), dilatation; and (3), contraction of the cavity of the organ.

The *hypertrophy* is a change which really deserves this title, inasmuch as it essentially consists of an exalted nutrition, and increased growth, of the muscular fibre-cells; and, in favourable specimens, is strictly limited to such a change. The calibre of the stomach being constricted by the tumour, an increased effort is required for the propulsion of its contents; and this addition to its function necessitates (and indeed brings about) an unusual development of its structure. The thickened muscular coat retains, however, its normal texture. Its areolar bundles certainly appear somewhat more distinct and larger than usual; but their size and strength, in proportion to the true contractile tissue, remain unchanged. The fibres themselves are perhaps rather redder and darker than normal; but even this alteration may be partially referred to the enlargement which their bundles have undergone.

The above purer form of hypertrophy closely resembles that which may often be seen in the muscular coat adjoining (and especially behind) the cicatrix of an ordinary gastric ulcer (p. 140). Like the latter, too, it may extend for a variable distance through the organ. It is usually limited to the neighbourhood of the pyloric region, which the cancerous deposit especially affects.

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[1] Apart from mere continuity of the adherent mass, which occurs very frequently; according to Dittrich, 22 times in 160 cases.

Many of its deficiencies and complications are explained by the circumstances under which they occur. In the softer varieties of cancer, and in tumours of rapid growth, such hypertrophy is generally either indistinct or absent. In instances where the deposit is traceable by no very abrupt line of demarcation into the unaffected part of the stomach, it is often less marked; or is accompanied by such thickening of the areolar dissepiments of the muscular bundles, as renders it difficult for the unassisted eye to discern how far the altered bulk and colour of these structures is due to mere hypertrophy, or to their implication in the cancerous disease. On the whole, a marked degree of this hypertrophy is rarely found, save in conjunction with a hard deposit, of slow growth, offering a tolerably abrupt edge towards the thickened muscular coat.[1]

Dilatation generally accompanies the above hypertrophy, and is very rarely found quite unmixed with it. Moderate degrees of either of these associated changes are difficult to verify, from reasons mentioned elsewhere.[2] But it is probable that in a large proportion of gastric cancers both these states are present. Still I think the proportion mentioned by Lebert (1 in 5) as evincing a "notable dilatation," must be partially referred to the variable estimates of anatomists respecting the average size of the stomach. For the cases I have collected would only furnish such an estimate by including in it many instances in which this organ was scarcely larger than a full meal would render it in perfect health. Of dilatation beyond this degree, the records of 214 cases afford me only 13 instances; a proportion equivalent to $6\frac{2}{3}$ per cent. In every one of them the pylorus was the seat of the tumour. And even with respect to these extreme instances, I think that it is rare for gastric cancer to bring about a degree of dilatation equalling that maximum of this state which is witnessed as the result of contraction in the cicatrix of a pyloric ulcer.[1]

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[1] On some of these details compare the remarks upon hypertrophy in the following Lecture.

[2] See remarks on this point in Lectures II. and V.

The *contraction* sometimes found in cancer of the stomach is seldom connected with any true hypertrophy of the muscular coat. It may be regarded as of two kinds; each depending on a different process; and each finding its parallel in another disease of the stomach. (See Lecture V.) In some cases it is the physical result of a specific pathological phenomenon: a slow shrinking or contraction of the scirrhous mass which occupies a large portion of the parietes of the stomach; constricting and diminishing its cavity in the same way as does the contracting tissue of that cirrhotic inflammation which generally attacks the organ with greater diffuseness. In other cases, in which the tumour occupies the cardiac orifice of the stomach, the stomach contracts (just as it sometimes does when an ulcer encircles the same aperture) simply because the constant regurgitation which this occlusion produces, prevents the cavity of the organ from undergoing its normal distension by receiving any quantity of contents. Here much of the contraction is temporary, and may be readily removed by artificial dilatation of the stomach. In some instances both these varieties of contraction are combined.

As regards its frequency, extreme contraction is far less common than dilatation. The 214 cases mentioned, only include 3 instances of contraction: of which 2 seem chiefly referable to the situation of the tumour at the cardiac aperture; and the remaining one to the contraction of a scirrhous mass engaging the greater part of the stomach.

The *ulceration* which generally engages the cancerous deposit has already been noticed; both as regards the local changes by which it is introduced, and the variable admixture of suppuration and sloughing by which it is often accompanied. Its remaining peculiarities need not detain us. That it is rarely or never arrested and repaired, the known features of cancer in general sufficiently inform us. That, as a rule, death intervenes before any large proportion of the gastric mucous membrane has been devastated by its extension, is equally explicable.

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[1] The accuracy of this opinion seems at first sight contradicted by my subsequent experience, in which large dilatation has occurred with about equal frequency in cancer and ulcer of the stomach. But the difference is probably referable to the larger proportion of such cancers which increased practice among the affluent (among whom gastric ulcer is certainly rarer than among hospital patients) has brought before me. And the *maximum* of dilatation still seems claimed by the ulcerous cases. (*Second Edition.*)

The *sequelæ* of this cancerous ulceration evince a marked contrast with those seen in ulcer of the stomach. Out of 507 cases of gastric cancer, in 21 *perforation* took place, with its usual result of peritonitis, rapidly ending in death. In 4 of these 21, however, the contents of the perforated stomach were not effused into the general cavity of the belly, but into an intermediate cavity which corresponds with the sac of the omentum, and was bounded by the adherent viscera that enclosed this sac. In 10 others, the action of perforation was shown to have been imminent by the necropsy; and had probably so far taken place, as to have allowed that leakage of the contents of the stomach, to which the fatal suppurative peritonitis seemed due. As regards *fistulous communications*, the above number includes one instance in which an abnormal opening of this kind led from the cancerous stomach to the anterior wall of the belly: one in which its cavity was thus thrown into communication with that of the jejunum; and no less than 11 in which the transverse colon was the seat of a similar aperture (twice by an intermediate cavity formed exclusively of cancerous deposit).

Each of these results is in striking contrast with its analogue in ulcer of the stomach. Thus in gastric cancer, perforation seems to be only one-half or one-third as frequent as in ulcer; its percentage being from 4 to 6 instead of 13. While conversely, the formation of a fistulous communication between the stomach and colon occurs far more frequently: how much I cannot definitely estimate, though I should assign it a proportion from six to ten times greater than in gastric ulcer.

This double contrast is in great part due to the peculiarities of that destructive process which occurs in the course of gastric cancer. Growth and decay, deposit and ulceration, are generally going on at one and the same time in different parts of the diseased mass. And thus, even at the very time that the sloughy or ulcerous surface by which the cancerous tumour abuts on the gastric cavity is hourly losing a certain proportion of its bulk, the opposite or peritoneal aspect of the tumour is rapidly throwing out a cell-growth which more or less replaces these ravages. Hence, even after long ulceration, the thickness of cancerous deposit between the cavity of the stomach and that of the peritoneum may still remain comparatively undiminished. No doubt this process may be regarded as to some extent paralleled by the deposit of lymph at the base and margin of a gastric ulcer; especially where (as is occasionally the case) the symptoms point to an uninterruptedly open state of the ulcer during a long period of time. But the analogy is a remote one. For while it is chiefly the situation and amount of such lymph which determine (p. 162) the occurrence or non-occurrence of perforation in the gastric ulcer, and such a quantity as is generally present in the ulcer of the posterior surface forms (in the majority of cases) an efficient barrier to this incident during an almost indefinite period, no amount of cancerous deposit can have any such protective efficacy. The mass it interposes may indeed, for the time, separate the gastric and abdominal cavities. But the protection temporarily afforded by its quantity is sure to be soon abolished by its quality. In other words, its cancerous nature shortly brings about an extension of the same softening or ulceration as that which already occupies the neighbouring mass, and the barrier gives way.

These circumstances are well illustrated by the fact, that the situation of the cancerous mass exercises no influence on the accident of perforation at all comparable with that seen in ulcer of the stomach. Indeed, from the posterior and diaphragmatic aspect of the cancerous stomach being the earliest and most frequent seat of adhesion, it is precisely in this situation (the safest a gastric ulcer can occupy) that the cancerous perforation most frequently takes place.

In both cancer and ulcer of the stomach, the occurrence of perforation, as a pathological event, is by no means synonymous with that characteristic and fatal group of symptoms which we generally associate with this word. Just as in the latter disease (p. 143) a true perforation of the gastric coats is often accomplished months (or even years) before an extension or renewal of the ulcerative process penetrates the new tissue which has hitherto warded off the accident; so in cancer of the stomach, that portion of this organ which corresponds to the diseased mass is often destroyed long before the destruction of the subsequent deposit brings about a communication between the gastric and abdominal cavities. But the degree in which any of the original structures of the part are left, it is sometimes impossible to define. The frequency of partial perforation—or rather of a leakage of the gastric contents through such a spongy mass—can scarcely be compared with that of the similar accident in ulcer. But its apparently greater frequency is readily explained by the above allusions. Lastly, in any strict comparison of the pathology of the two diseases with respect to this accident, the date and mode of death ought not to be overlooked. It may fairly be conjectured that a much larger proportion of gastric cancer would end in perforation, were it not that the collateral circumstances of the disease often destroy life before the local mischief has reached this stage of development.[1]

It is not easy to offer any hypothesis for the relative frequency of communication between the cancerous stomach and the colon. The selection of this intestine is of course explained chiefly by its situation. But while the above characters of the process of cancerous deposit no doubt constitute the main cause of this particular variety of perforation, as well as of the accident in general, the fact that this one variety is as frequent as all the others put together, suggests some peculiarity, favouring either the deposit or the removal of cancerous substance, and connected with the colon, rather than with the various other structures which adjoin the stomach. Perhaps, however, the mere thinness of the intestinal coats, as contrasted with these structures, will account for its being more rapidly and frequently penetrated when adhesion has once taken place. At any rate we can scarcely at present assume any specific liability of the colon

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[1] For example, if we may regard the establishment of an unnatural opening between the cancerous uterus and the bladder or rectum, as analogous to perforation of the peritoneal cavity by a cancerous stomach, it may be interesting to notice that there is good ground for estimating the former accident to be from two to four times more frequent than the latter, in equal numbers of cases of gastric and uterine cancer.

(in virtue of its structure or function) to an accident of which the more immediate conditions seem so evidently local.

The *hæmorrhage* which occurs in the course of gastric cancer affords, in some of its varieties, an equal contrast with that witnessed in gastric ulcer. As a rule, it only occurs after the access of ulceration; though, prior to this event, it may be produced by mere passive or active congestion—a form of bleeding which, from obvious reasons, seems to be much more frequent in cancer than in ulcer. The exact frequency of moderate hæmorrhage can scarcely be estimated in either malady. But those larger bleedings which occur as a result of the lesion of a considerable artery seem to be much rarer in cancer than in ulcer. Out of 374 cases, only 4 exhibit such a hæmorrhage: a proportion of barely more than 1 per cent., or one-fifth of that estimated for gastric ulcer. As might be expected from the usual situation of the cancerous deposit, all of these appear to have been lesions of the superior pyloric artery (p. 20).

As regards that complete obsolescence of the cancerous deposit which is evidenced by its conversion into cretaceous matter, the 214 cases I have collected only afford one instance of this kind—a woman, aged thirty, affected with colloid cancer of the pylorus, the liver being also occupied by medullary deposit. A similar instance described by Dittrich[1] suggests equal doubt how far this process really deserves the above name. While these two cases suffice to modify Rokitansky's opinion—that it is the hard and fibrous cancer which is exclusively amenable to this process—their rarity reduces them to a very exceptional variety of the disease.

In respect to the healing of cancer, the dead-house scarcely modifies the gloomy opinion derived from clinical experience. In every instance hitherto brought under my notice, I have assured myself that the gastric cicatrices which suggested a healed cancer were precisely similar to those of ordinary ulcers. Of course such a statement does not invalidate the observations of others, who have found scars cover a scanty scirrhous deposit. But it is possible that some of these instances have been deposits of scirrhus in the cicatrices of ordinary ulcers; and that in others, the fusiform cells and fibres of such scars have been mistaken for those of scirrhus, from which they are sometimes scarcely distinguishable by the most sedulous examination. It is certain that few or none of these cases are authenticated (as they certainly should be to establish so striking a fact) by a careful comparison with the symptoms of the patient during life. Lastly, I fear that it is more than unsafe to accept statements which (like Lebert's) allude to these appearances in language so equivocal as the term "scars of cancerous ulcers" seems to be, when brought forward by a pathologist whose clinical research, however assiduous, does not enable him to depict so common a disease as ulcer of the stomach from his own experience.[1]

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[1] *Loc. cit.*, p. 15.

The *ætiology* of cancer of the stomach has so little direct connexion with the symptoms which attend the malady during life, that I now only need sum up those pathological details which, from their prominence and constancy, seem most to suggest a causative relation. The most obvious (at any rate the most convenient) hypothesis of the disease would refer it to a causation, which is probably itself the co-efficient of at least two elements: the disease and its site; the cancer, and the stomach it invades.

In respect to the former of these two elements of causation, there are no facts which entitle us to suppose that the disease (whether humoral or not) presents any specific modifications in the stomach. At any rate, the circumstances of age, sex, &c., already noticed, afford little countenance to such a supposition.

Assuming so much of the ordinary views respecting the cancerous diathesis, as to infer that its intensity is expressed and measured by the cancerous deposit with tolerably equal accuracy in all the organs generally affected by cancer, it is chiefly as to the selection of the stomach by this deposit that we may examine into the facts now brought together.

We have successively seen that, in a large proportion of cases, the disease selects the stomach; that in this organ itself, it further chooses out the cardiac and pyloric orifices, and especially the pyloric. Its situation, in the earliest stages in which we detect it, conclusively shows that it cannot be attributed to any mechanical or chemical effects of the ingesta; that it is not due to any change in the secretory apparatus of the stomach; or even to a lesion of any part of the mucous membrane, or of the tissues immediately subjacent. In short, that, for practical purposes, the deposit must be regarded as seated in the loose sub-mucous areolar tissue of the stomach, at some little distance from the active cell-growth of the gastric surface; and generally, so much nearer (or more closely allied) to the similar connective tissue between the bundles of unstriped fibre, as to involve these long before reaching the mucous membrane.

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[1] *Loc. cit.*, p. 526.

The organ thus affected is, with the exception of the rectum, the œsophagus, and the uterus, the thickest and strongest mass of unstriped muscle in the human body. Relatively to its function, indeed, it transcends the contractile structures of all three of these organs: because that function implies constant and protracted movement, instead of such an intermittent and brief contraction as that by which all of them might readily be shown to impel their contents. How violent, as well as protracted, that gastric movement is, may be easily conceived when we recollect that, for about six hours of the twenty-four, the stomach is actively contracting upon (and propelling) its contents; by a movement which, during a great part of this period, almost obliterates the cavity of the pyloric half of the organ every two or three minutes.[1] Hence, apart from its mere bulk, we may fairly suppose that the muscular coat possesses an exalted nutrition, a rapidity of growth and decay, which proportionally exceeds that of either of the other masses of unstriped fibre with which we have contrasted it.

Should future researches establish, either the absolute commencement of the disease in the unstriped fibre-cells themselves, or (what seems more probable) an exactly analogous situation of its development in all four of these organs—stomach, uterus, œsophagus, and rectum—of course the above conjecture would acquire a somewhat firmer basis. In any case we must recollect, that the areolar tissue enclosing and penetrating any muscular mass necessarily shares in that mechanical displacement which this muscular mass executes (and often, from its very office, is more suddenly and violently displaced than the contractile fibres themselves); and therefore as necessarily becomes the seat of a nutrition, exalted beyond that of the same tissue in less active parts.

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[1] Compare p. 12.

It is therefore to the more energetic movement of the pyloric half of the stomach, and to the passive (as well as active) relations of the pyloric and cardiac valves at the extremities of the organ, that the frequent selection of these parts by the cancerous deposit may be directly or indirectly ascribed. Whether the peculiar structure of the organic muscle, as a comparatively permanent cell-growth, invites the access of a disease the morphology of which is closely akin to its own—is a question beyond our present knowledge to decide, and little affecting those gastric peculiarities to which we are now limiting our attention.

Such an hypothesis, however, as that we have advanced, ought never to be stated without an exposure of its chief deficiencies; a knowledge of which restrains any conjecture to the useful office of grouping facts, and at the same time prepares for its confirmation or rejection. While it is not impossible, for instance, that the conditionating cause (*causa causativa*) of the preference of this or that particular organ by cancerous disease, may be a different (or even compound) one in each, it is difficult to fit into such an hypothesis as the above the frequency of cancer of the uterus and mamma. In the former organ, we may perhaps shadow out something compatible with it in that remarkable activity of growth and decay which the muscular uterine wall from time to time undergoes; connected as the disease is with a period of life, when we might readily imagine an accumulation of nutritional activity, or even a transfer of a tendency to growth from one part of the reproductive apparatus of the female to another. But in the latter organ, this vague formula of transferred activity is alone left us; and—if we except the equivocal influence of mechanical violence in producing cancerous tumours of the external parts, and the more marked effect of cutaneous irritation on cancer of the lip and scrotum—it is difficult to trace any community of causation between the gastric and mammary lesions. The conjecture hazarded with respect to cancer of the stomach therefore remains doubtful;—confirmed by facts themselves requiring investigation; and opposed (though not contradicted) by some of the best evidence which can at present be adduced.

TREATMENT.

The treatment of cancer of the stomach might almost be left unnoticed. Not merely because we are hopeless of its effecting the cure of the patient. For the alleviation of sufferings which cannot be removed, and the postponement of a death which cannot be escaped, are too often the only benefits to be expected from the application of the Art of Medicine to disease in particular, as well as in general. But rather because the conditions through which this terrible disease conducts the sufferer towards his death are too secondary and collateral to the malady itself, to admit of such a common plan of treatment, as, with suitable modifications, is often applicable to the numerous varieties of a given disease.

Hence I shall only hint at the principles which ought to guide our remedial attempts; and shall refer you, for the means of carrying out those principles, to what is elsewhere (Lectures II. III.), said respecting the treatment of the diseases which are imitated by the secondary results of gastric cancer.

Looking back at my own experience, I must say that I have done more good by careful feeding than by any drugs in the Pharmacopœia. These cases, however, have been chiefly among Hospital patients. In other words, they were instances in which the charity of the unprofessional public made me the means of suspending that destitution and wretchedness by which cancer can be produced, and aggravated, in the strictest pathological sense of these words. Pray acquit me of exaggeration if I say that, more than once, as certainly as I have seen vaccination reproduce cow-pox, I have witnessed misery, anxiety, and starvation, suddenly rouse into fatal activity a gastric cancer: and that, this cancer having run its course in a few weeks, the necropsy has verified anatomical details conclusively proving that it must have been present as an unsuspected tumour for months, or even years, during which the patient has literally enjoyed the most robust health. And even in a case of this kind, I have known agony and prostration converted into comparative ease and cheerfulness during the patient's last few weeks of life, by the comforts of an Hospital: among which I especially distinguish light, air, warmth, stimulants, and food of proper quantity and quality.

Perhaps in some instances we may suspect a still more specific reason for the remarkable improvement evidently procured by careful nourishment. As you will hear in the next Lecture, muscular hypertrophy of the stomach is one of those curative efforts by which Nature partially and temporarily counteracts the obstruction producible by gastric cancer. Now since, on the one hand, the nutrition of every organ depends upon that of the whole body, while, on the other, this secondary obstruction sometimes dictates the whole of the gastric symptoms present, that group of symptoms which in every case forms (clinically speaking) the disease, may be relieved (and even removed) by suitable measures of this kind.

But while a generous diet is in most cases indicated, alike by the nature of the lesion, and by the age and circumstances of the patient, its use is generally forbidden by the state of the stomach. That complete failure of the appetite, which often forms an early feature of the malady, also opposes what is practically a great obstacle to a diet of this kind. While the pain, distension, and vomiting which are present in the more advanced stage, constitute hindrances such as the physician is even less likely to vanquish. To vary the food, in what is often a fruitless search after some nourishing articles of diet which (guided by the patient's instinct) we fancy the stomach may bear; to give these in small and frequent meals; and (especially where the stomach will support no other stimulants) to administer opium;—such is often all that we can accomplish. Of course diarrhœa, constipation, and excessive pain suggest their appropriate remedies; which in the two former, are often best given as enemata.

But in all these points the treatment of gastric cancer is often necessarily akin to that of gastric ulcer. Indeed, we have seen that its later stages (and sometimes even its earlier symptoms) are associated with such an ulcerated state of the surface of the cancerous tumour, as quite accounts for the indications of its treatment being almost identical with those of gastric ulcer. Hence I may refer to the measures recommended for the cure of the latter disease in the preceding Lecture; merely premising that, on the whole, wine and other restoratives may be given, not only with fewer scruples, but with more advantage.

The *prognosis* can of course only vary in respect to the date at which it tends to fix the fatal termination of the disease. And in our conjectures on this point (which is often very important in practice) the treatment rendered necessary by the peculiar features of the case is scarcely less influential than the various pathological occurrences already dwelt upon. Much as the fatal event may be hastened by intense and continuous pain, by excessive vomiting, by frequent hæmorrhage, by a rapid growth of the tumour, by secondary deposits of cancer elsewhere, and a variety of other circumstances of this kind; and obviously as œdema, emaciation, prostration, or delirium, often constitute, not so much forebodings, as "the beginning of the end;" still the inability to take food is sometimes a source of at least equally imminent danger: such as (for example) may in a few days hurry to the grave a young and hitherto well-nourished patient, in whom the absence of all these symptoms or complications in any marked degree might otherwise lead us to expect the malady would extend to its ordinary duration of a few months. And as it is not always easy to say how much food is really taken, or retained, by the patient, this rule of prognosis is practically by no means the truism which at first sight it seems to be.

LECTURE V.

Cirrhotic Inflammation, or Plastic Linitis of the Stomach—Suppurative Linitis—Tumours—Hypertrophy—Atrophy—Dilatation; from obstruction, destruction, injury, paralysis—Secondary Inflammation.

In attempting to sum up the chief features of several diseases of the stomach by the following brief discourse, I may premise that the maladies to which it refers, though exceptional in one sense, are not so in another. For even the rarest among them are not (at least in my judgment) the mere extremes or modifications of a variety of diseases: but rather constitute types and classes for themselves. And not only are they of great pathological interest, but their collected cases are quite numerous enough to afford deductions of much practical importance in the treatment of those more common gastric maladies which are allied to them. I may add, that my remarks are all founded on clinical and pathological observations; and that I hope they throw some new light upon the maladies of which they treat;—maladies, some of which are not only obscure in their nature (and therefore little likely to reveal themselves to the casual or one-sided glimpses which their infrequency has caused to be bestowed upon them), but even obscured by the names and descriptions they have hitherto received.

CIRRHOTIC INFLAMMATION, OR PLASTIC LINITIS.

The first of these diseases is one which in its less marked forms is not by any means unfrequent. But from the obscurity of its symptoms, and the closeness of their resemblance to cancer, we may usefully reverse the ordinary course of description; and discuss successively its appearances, its nature, and its name, before glancing at its diagnosis and its treatment.

We shall first notice what I venture to call typical specimens of the lesion. In such cases, the necropsy of some obscure gastric disease of long standing shows a stomach which, even on our dividing the wall of the belly, strikes us as remarkably altered. Sometimes large, sometimes small, perhaps of average dimensions, it has a peculiar pearly whiteness and opacity; an appearance which is partially due to a dulness of the peritoneal coat, in remarkable contrast with its ordinary mirror-like brilliancy. Removing the organ for further examination, we find this change in its colour associated with a great increase in its weight and density: so that, for example, it has a hard, gristly feel; and not only fails to collapse by its own weight, but resists a considerable pressure; or returns to its original shape on the removal of such pressure, like a large artery, or a caoutchouc bottle. An incision through the coats of the organ exhibits a vast increase of its thickness: a change which, as it is diffused equally throughout the stomach, leaves the relative depth of vertical sections at different parts little affected; and is hence summed up by the statement, that the thickness is some six or eight times greater than what is normal. But in spite of this thickening (which is often attended by an increased density that makes the pearly-white section fairly creak while it is being traversed by the scalpel), the different gastric tissues remain not only discernible, but distinct from each other. Often, indeed, their adjacent boundaries are evidently composed of a looser tissue than that of the mass elsewhere; while even the proportionate thickness of the areolar, muscular, and serous coats sometimes approaches that of the healthy tissues. The mucous coat, however, appears much less affected; its matrix being either little changed in thickness, or indistinguishably fused into the subjacent "tunica nervea," and its secretory structures remaining substantially healthy. Finally, the whole organ is comparatively bloodless: a condition which is not only in marked contrast with its normal state after death (compare Lecture II.), but is, of course, most remarkable in the more vascular of its coats; to wit, the mucous membrane.

A closer inspection of such a specimen shows that the abnormal character is not limited to a mere increase in thickness. However distinct the gastric coats, they are far from offering that contrast to each other which is characteristic of their healthy structure. Muscle, areolar tissue, matrix, and mucous membrane—are all unnaturally alike. All evidently owe their increased thickness to the presence of the same new substance; the uniform and interstitial deposit of which is one of the main characteristics of this gastric lesion.

The examination of this deposit under the microscope confirms the impression which it affords to the unassisted eye. Aided by dissection, the latter discovers that the white mass consists of a tough, semi-elastic, imperfectly fibrous material, closely analogous to that found in common fibrous tumours, and in the indurated cicatrix or margin of a common gastric ulcer. The microscope does but follow this conclusion into details, by showing (mixed with the original tissues in variable quantities and conditions) a mass of a rudimentary fibrous character:—composed, that is, of a substance, the wavy filamentous markings of which are less distinct than in the white fibre of ordinary areolar tissue; from which, too, they are further distinguished by the scarcity of vessels, the absence of yellow or elastic fibres, and the presence of scattered cytoblasts and nuclei. Developmentally, these nuclei seem to represent in this new structure, the elastic fibres of the normal tissue: while functionally, they may be conjectured to afford at least a partial compensation for the absence of vessels; the nutritive uses of which they replace much as do the cartilage-cells of those tendons which play over bones. Some of these nuclei occasionally surpass the stage of development to which the latter (their normal analogues) are restricted; or rather, deviate from it altogether, so as to be traceable, not as mere elongated nuclei, but as fusiform cells of variable (usually small) size. The direction of these fibres appears to be very irregular; a tangled interlacement in various senses, rather than any special or systematic decussation.

A careful comparison of the several coats of the stomach shows that they are engaged by the deposit in very different amounts, and with still more different results. As regards mere thickening, the areolar tissue of the "tunica nervea" is most altered; its depth, as seen in a vertical section, being often increased from ten to twenty-fold. Next to this comes the peritoneum: it (or rather its sub-serous areolar tissue), acquiring a thickness which, though rarely exceeding a fourth of the preceding sub-mucous tissue, is, proportionally to its original tenuity, sometimes almost as great an increase; or a multiple of seven to ten times the natural bulk. The muscular coat ranges from five to eight times its pristine thickness; and the mucous membrane (in which term the matrix ought to be included) is scarcely ever more than doubled or trebled: proportions which we shall see are often not exclusively due to the new deposit.

Rarely do we find either of these tunics merely increased by the new deposit, without any degeneration or decrease of its original tissues. And hence each of the above proportions must be regarded as the difference of two processes in addition and subtraction, or as the degree in which the former outweighs the latter, rather than as a mere increment to the corresponding normal structures. And while the degree of these antagonist processes is liable to great variety, the following summary may be offered of their course in the several tissues.

The *serous* coat is almost always damaged, or rather destroyed, over a great extent of its surface; the epithelium either failing altogether, or being replaced by an extremely irregular and scanty cell-growth, very unlike its beautiful tesselation in health. And while its sub-serous tissue is generally solidified by the deposit, which strangles and kills its original vessels and fibres, nothing is commoner than to find decisive evidence of superficial inflammation, in the shape of recent pasty lymph, or older threads and fibres, gluing the stomach to adjacent viscera, and having a structure no way differing from that of such adhesions in other gastric diseases. Occasionally these appearances of peritoneal inflammation extend to the neighbouring duodenum.

The *mucous* coat is, in general, much less seriously involved. Often, but for its thickened matrix, it might almost be thought healthy. But while its pallor of surface, and the white lines running upward between its tubes, show how seriously its circulation is interfered with by the mere change of its matrix, an increased degree of this deposition readily inaugurates further lesions. Thus, here and there, the tubes become distorted from their proper direction, or even destroyed in some (usually the lower) part of their length. Sometimes a similar (but apparently more sudden) mechanical interference with their vessels gives rise to appearances of local extravasation or gangrene. While the loss by ulceration of a variable extent of the whole thickness of this coat is so frequent and dangerous a result of this kind, as really to constitute what might almost be called a termination or event of the malady.

Another curious change, belonging equally to the mucous and sub-mucous coats, may be mentioned here; namely, a kind of folding or plaiting of a variable extent of the mucous membrane, so as to raise it above the neighbouring gastric surface. Some of these cases are really very little more than a deposit in the sub-mucous tissue : a deposit which, by obliterating and filling its meshes, destroys their function, and thus fixes and renders quite ineffaceable the normal *rugæ* (p. 14) of the affected portion of mucous membrane. In other cases, however, it is the matrix of the mucous coat which is chiefly concerned : and in proportion as this is the case, the raised and wrinkled surface becomes a more complex and definite excrescence, in which the tubes are sure to be distorted and obliterated to a considerable degree. Even here, however, the low elevations of the membrane are seen, in a vertical section, to possess precisely the same structure as the adjacent healthier parts ; and the distinction of the original tissues remains well marked. In short, the change looks more like (what it doubtless is) a folding of mucous membrane upon and around a subjacent contraction, than that interstitial enlargement of a true cauliflower excrescence, which it distantly suggests and resembles.

The thickened *muscular* coat offers changes of even greater pathological interest than the foregoing. In a vertical section, the space belonging to this coat constitutes a straight uniform interval between the parallel lines of the dense white deposit in the sub-mucous and sub-serous areolar tissues. If the vertical section be parallel to the long axis of the stomach, we see a number of thick white threads, which pass to and fro between these two surfaces of the continuous deposit, giving and receiving a branch here and there, so as to form a kind of network, the large meshes of which enclose a substance of a gelatinous, brownish appearance. These dissepiments are evidently the thickened and enlarged representatives of those processes, which originally pass between the above strata of areolar tissue, and limit and ensheathe the bundles (p. 7) into which the fibre-cells are aggregated in the transverse layer of the muscular tunic. The microscope shows them to contain, with new substance, a great deal of this original tissue. And this view of their nature is confirmed by the simple expedient of varying the above vertical section for one at right angles to the axis of the stomach, when the muscular bundles being taken in a direction parallel to their course, the marked character of the fibrous network by which they are isolated almost disappears. The same expedient allows of an inspection, which shows that the brownish mass occupying the cells of the honeycombed longitudinal section is altogether made up of the proper muscular fibres. The fibres themselves are sometimes distorted : occasionally shrunken and diminished in size. But they are oftener enlarged : though not (I believe) to such a degree as quite enables us to dispense with the supposition of an increase in their numbers, to which the increased thickness of their mass is partially due. The longitudinal layer of the original muscular coat can rarely be traced : a disappearance which seems explicable by its original tenuity, and by its arrangement relatively to the sub-serous and transverse layers above and beneath it, without supposing it to undergo any peculiar absorption.

Now, just as it is impossible to question the nature of the muscular thickening above described, so there can scarcely be any doubt as to the origin of such a genuine hypertrophy of these unstriped fibres. We find a deposit which works the most serious (and despite their slowness, destructive) changes in every other gastric tunic, bringing about a positive increase and enlargement of the muscular tissue; and that, too, in a degree closely, if not exactly, proportionate to its original bulk in different parts of the stomach. We shall presently see that it is not always that this deposit is followed by such a muscular hypertrophy. Hence its causative influence is probably indirect: and can only be found in that increase of the contraction, and therefore of the nutrition, of the muscle, which the obstructive effect of the deposit itself evokes. It is the same true hypertrophy of an overworked muscle, which we see in the blacksmith's arm, in the dancer's leg, or (a closer analogy) in the cardiac hypertrophy of an obstructed aorta.

Among the variations from the above type of this lesion, we may first notice the existence of irregular lumps or patches; formed by portions of deposit, harder and more condensed than elsewhere. As might be expected, such aggravations of the general lesion are most frequent in the sub-mucous, and next to this in the sub-serous, tissue.

Contraction and *dilatation* of the stomach are two other variations from the type we have selected. Of these two opposite changes, contraction is not only by far the more common, but seems to be contrasted with dilatation in its constituting, not merely an exceptional or casual effect of the peculiar circumstances of any given case, but a law in the progress of the malady. Some of the appearances already described imply at least a partial or local effect of this kind. And though we have, for descriptive[1] purposes, selected a type of the lesion such as stands midway between the above two converse processes, yet a consideration of its characters, as just enumerated, points definitely to a more general contraction; which begins at an early date of the deposit, and continues steadily to increase (if not restrained or over-matched by the causes of dilatation) throughout a long period. In cases of long standing, the organ is thus gradually reduced in size, and often conversely increased in the thickness of its coats, until perhaps its cavity at last dwindles to a capacity of five or six cubic inches, and its coats acquire an inch of thickness. But this effect is more frequently interrupted long before its attaining such an extreme degree: and the stomach appears to remain many months or years[1] with little further alteration in this respect. Hardness, however, seems to increase up to the last.

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[1] Because where contraction of this kind exists, it must of course be allowed for in estimating the increased thickness of such an organ, and especially of its muscular coat (compare p. 72). And to do this with accuracy is difficult.

The much rarer incident of dilatation appears to be connected chiefly with two circumstances;—a rapid and extensive deposition of the new substance; and an enfeebled or degenerate state of the muscular coat. Of these two the latter may well be caused by the former. The dilatation is rarely excessive: indeed, it seems to consist rather in the thickened organ being fixed at its largest healthy dimensions, than in a dilatation such as is sometimes brought about by a cicatrized ulcer. The stomach is incontractible, rather than really dilated.

On the foregoing analysis of the various records and preparations of this lesion we may found some plausible conjectures respecting its nature; at any rate in this its typical form.

As regards its morbid anatomy, it is evidently an exsudation, occupying the areolar tissue of the stomach, and gradually undergoing a change, in which its development into a low grade of fibrous tissue (like that of a fibroid tumour) is accompanied by a constant decrease of its bulk, and increase of its density. The contraction and hardening thus brought about not only seriously damage the function of an organ to which mobility is essential; but they obstruct its circulation, and probably its innervation also; and they inaugurate grave lesions of its mucous and serous coats. Even in the muscular tunic a similar effect sometimes obtains. And though, incidentally, the obstacle formed by the exsudation often evokes, in this powerful organ, a change which diminishes and defers its disorganization, and even exalts its nutrition for the time; still in the long run, this salutary effect generally yields to the interference produced by the slower but surer agent—to the contracting and hardening of the interstitial deposit. The physical effect transcends the physiological effort: the hare is caught by the tortoise, and then distanced in the race.

But what is the pathology of the lesion? Regarding it as (what it obviously is) both inflammatory and gastric, shall we therefore name it *gastritis;* adding (it may be suggested) the term *interstitial*, to distinguish it from inflammation of the mucous membrane? Or, looking to the evidently exalted nutrition and vigour of the muscle, and the thickening of the areolar tissue by a structure somewhat analogous to itself, shall we therefore call it *hypertrophy?* Or shall we look to its hardness to supply us with a name—*sclerosis?* Or find a more characteristic title in the histology and arrangement of the deposit—*fibroid infiltration?*

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[1] To judge by the tumour perceptible in the epigastrium during these periods.

Now, though names are even more important in Pathology than in some other sciences (simply because they exercise even more than their customary influence on ideas), it would scarcely be right successively to question the applicability of each of these terms to this lesion, unless for the sake of the inquiry, as well as of its results. But to call such a lesion "gastritis" is almost as objectionable as it would be to call pleurisy, pneumonitis. For the latter termination of the name of an organ ought to be reserved for inflammation more or less specially involving its peculiar structure—that structure which secretes gastric juice in the stomach, or which interchanges gases with the air in the lung. While the involvement of the gastric mucous membrane in this disease is inconstant, late, and incidental. Then, again, "hypertrophy" is clearly a misnomer, both as regards the whole organ, and the areolar tissue; portions of the original mass of which may often be found atrophied and decayed in the new deposit that entangles and surrounds them. While, even in respect to the muscular coat, we have seen that its genuine hypertrophy is not a real element of the disease, but an attempt of Nature (so to speak) to remedy or palliate the effects of the lesion: an attempt which appears to be sometimes wanting, and generally temporary. "Sclerosis" we need not criticize: since, apart from its applying chiefly to the later stages of the lesion, its etymology affords no sufficient distinction, either from scirrhus, or from calcified deposits. "Fibroid infiltration" is less objectionable than any of the preceding. But the adjective overstrains an analogy: the substantive misstates a fact. For the histology and progress of the deposit is very unlike fibroid. While not only do we never see it as an infiltration, but what we do see by no means looks like the solidification of the liquid deposit this word ought to connote: inasmuch as it differentiates the various tissues, instead of soaking into all in tolerably equal amount.[1]

In point of fact, while this inflammatory lesion is, in many of its details, quite unlike any other known to our existing Pathology, it has two analogues which specially deserve notice: one as merely offering an interesting (though imperfect) resemblance; the other apparently the result of a pathological relation close enough to dictate the extension of its own name to the more obscure and infrequent gastric malady. Some of the varieties of what is known under the name of "phlegmasia dolens" bring about a condition which may be regarded (*mutatis mutandis*) as ultimately closely akin to certain stages of this disease of the stomach. But though it is scarcely[2] impossible, it does at present seem improbable, that an obstructive phlebitis of this organ generally inaugurates the lesion. Its other and closer analogue is found in cirrhosis of the liver. In this disease we may trace so close a resemblance to the lesion under discussion, and a resemblance which extends to so many circumstances of the deposit—its nature, its situation, its relation to the vessels, its connexion with the portal system, its contraction, its effects on the adjacent original structures—that really the application of the term *cirrhosis of the stomach* seems by far the best means of connecting its gastric analogue with our existing nosology.[3]

But these typical cases are so rare, that, in spite of their great scientific interest, I should scarcely feel justified in giving them a special description among the maladies of the stomach, were it not for their extrinsic claims to clinical importance. It is because there is a class of gastric lesions, which occur with comparative frequency, and of which the preceding are the best illustrations (if not representatives), that I have ventured to depict a lesion which you may perhaps never see twice in an extensive practice.

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[1] It would be easy to show that the analogy suggested above (between this malady and pleurisy) is rendered more exact by the close physiological relation between serous membranes and areolar tissue. (Compare "Cyclopædia of Anatomy," article "Serous Membranes," by the author.)

[2] The actual concurrence of phlebitis in other parts of the body has been noticed in one or two cases.

[3] In saying this, I imply no opinion as to the goodness of the term "cirrhosis" itself. On the contrary, like "cyanosis," (which, to mean anything, ought to include such dissimilar maladies as bronchitis, heart-disease, and Asiatic cholera,) it connotes, not the lesion, but one of its separable accidents. And although any new name is likely to be merely provisional to future pathological discoveries, I would suggest that the inflammation of the filamentous network of areolar tissue ensheathing the vessels, which seems the main characteristic of both lesions, might be well expressed by some such word as *linitis* (from the Homeric λίνον, *rete ex lino factum*).

Thus it is common enough in the pathology of the stomach, to find a condition which is at least closely akin to this universal cirrhosis, occupying the pyloric half or third of the organ. All gradations, too, between this extent, and the total involvement of the stomach, can be found in the collated history of a large number of specimens. And since you will recollect that even in this total involvement, this lesion generally increases in thickness up to the pyloric valve, these gradations may fairly be connected with a presumption in favour of the identity of the two: the more so that, on the whole, the lesion appears to be increasingly frequent in proportion to the closeness of its limitation to the pylorus. But from whatever cause, the gradations between the two, though scarcely deficient, do not seem to be uniformly filled up, being rarer than the universal cirrhosis itself.

The microscopic examination of marked specimens of this kind confirms the above view of their real identity with the preceding. The various tissues of the pyloric sac are still quite distinct from each other; and the deposit still involves the same structures, in much the same proportions. Histologically, too, it also is evidently a low fibrous tissue, devoid of the copious cell-growth rarely absent from true cancerous deposits. Its influence on the muscular coat equally resembles that of the general cirrhosis. The hypertrophied transverse layer thickens rather more rapidly up to the pyloric valve: but with the slight exception thus implied, its serous and mucous aspects are parallel; and its network of hypertrophied dissepiments encloses a similar brownish mass of unstriped muscular fibres.

The serous and mucous membranes are not affected quite in the same degree as in the general lesion. In the former tunic, inflammation is much less frequent, and even the dull, rough, pearly appearance already mentioned is less constant and characteristic. In the mucous membrane, again, ulcerations seem less frequent, as are also the irregular elevations of its surface, as well as the distortions of its stomach-tubes they produce. But these diminished effects of the deposit on the mucous and serous membranes appear to be accompanied by a tendency to its greater (rather than less) aggregation in the sub-mucous areolar tissue itself; where it often has a vertical depth disproportionately exceeding its limited lateral extent. Even here, however, the looser and more distinct stratum (*e*, *f*, fig. 6, p. 14), of areolar tissue which adjoins the true matrix (p. 17), sometimes suggests the centre of this areolar layer to be the chief seat of the deposit.

But as regards the nature of these appearances, it would be begging the question very reprehensibly to omit mentioning, that the specimens thus classified as a more frequent (though less typical) variety of cirrhosis of the stomach, and described as due to a non-malignant fibroid deposit in its coats, are still regarded by good authorities as cancers;—in fairness, I think we must say, as incipient cancers. And the chief arguments for and against this conclusion deserve notice; not only because they involve one of the most obscure and important points in the pathology of the stomach, but also because the facts which sustain these arguments incidentally elucidate the pathological nature and relations of the deposit, whether cancerous or otherwise. They may, I think, be tolerably summed up as follows.

In favour of their cancerous nature are these facts. The age of their subjects; which closely corresponds with that period of life most subject to gastric cancer.[1] Their situation in the stomach—namely, their marked predilection for the pyloric region, which is their chief (often their only) seat; as well as the origin and preponderance of their deposits in the very same stratum of sub-mucous areolar tissue in which we have already traced the ordinary development of true cancer. To these constant features of resemblance accede others less regular and less frequent.

Thus in form, the two deposits often approximate to each

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[1] Here I am alluding, not to a calculated (see p. 133) liability—for these cases are not numerous enough to justify such an estimate—but merely to the average age of their subjects.

other; the cancerous sometimes affecting a flat or lateral extension; the cirrhotic (as already noticed) a tumorous or vertical. The manner in which both affect the different gastric tunics is also sometimes very similar. As respects the mucous membrane, the low irregular elevations of the fibroid disease are sometimes closely imitated by a cancerous deposit in or near the deeper surface of the matrix; just as, conversely, an unusual prominence of these parts of the mucous membrane suggests and simulates cancer. The serous membrane is often the subject of an analogous (if not identical) inflammation in both deposits. And lastly, as regards the muscular coat, the distinctions between cancer and cirrhosis, often marked enough, are so frequently obscured by similar gradations, that the question really becomes one of degree rather than of kind. As a rule, the muscular coat of the cancerous stomach is doubtless displaced, distorted, or even destroyed, far more frequently than that of the cirrhotic organ or locality. But, on the other hand, as already specified (p. 251), interstitial hypertrophy of the muscular substance is anything but uncommon; and, in exceptional cases, is quite regular and even enough (both in its thickness, and in its diffusion over the affected segment of stomach) to be scarcely distinguishable from the hypertrophy produced by cirrhosis. And it must be remembered that in the latter lesion, not merely the hypertrophy, but the very structure which forms its seat, is often gradually removed by the further changes of the deposit. Lastly, even the presence or absence of the cell-growth usually characteristic of cancer, is not always adducible as evidence for or against the malignant nature of the deposit. For, on the one hand, such cells are sometimes detected in but moderate (or even scanty) numbers in cases the true nature of which is unequivocally shown by the presence of secondary cancerous deposits in other organs:—so that, for example, the sedulous examination of a hardened pylorus sometimes reveals nothing suggestive of cancer; while the adjacent lymphatic glands, the liver, or the lungs are occupied by masses of unmistakeable medullary growth. On the other hand, the developmental cells of the cirrhotic fibres are sometimes to be met with in considerable quantity, and of a form which resembles that of the fusiform cells of a scirrhous deposit.

Before stating the reasons against such an identity, I venture to eliminate a good many facts of the above kind; on the ground that the distinctness of the general characters of two diseases is not much affected by the circumstance that examples are sometimes met with equally referable to both. Whether two diseases are distinct, is one question: whether we can always distinguish them, is another. Nobody doubts the difference of the two sexes, because we sometimes meet with an instance in which neither its aspect during life, nor its dissection after death, reveals the true (or even preponderant) sex of an hermaphrodite animal. Nor need we trouble ourselves with arguments which, if good for anything, would identify a fibroid with a cancerous tumour of the breast; and confound gastric ulcer with the gastric cancer from which Cruveilhier and others distinguished and withdrew it.

It is more to the purpose to point out, that some of the above resemblances are quite insufficient to suggest any real identity. Thus, whatever the general similarity in the age chiefly affected by the two lesions, still (like the analogous similarity in the case of gastric ulcer and cancer) it cannot be followed into details. For while we have found the average age of the subjects of gastric cancer to be about 50 (p. 223), that of these cases of cirrhosis is about 34. The general selection of the pylorus as the chief or exclusive site of the deposit is almost as unlike the exact preferences of situation shown by cancer, as are the analogous predilections (so to speak) of gastric ulcer. And in a majority of cases, others of these resemblances so far contradict each other, that the distinctness of the cirrhotic lesion in any given instance is far less difficult than their mere enumeration would suggest. In general, the decision turns upon facts by no means difficult to detect or interpret. When we find a lesion, which has been perceptibly present for several years, and in spite of this (compare p. 271) duration, and of a considerable bulk, has yet failed to fuse the coats of the stomach together; which leaves intact the muscular (if not the mucous) tissue; which extends laterally, but not vertically, in and between these coats, so as to thicken a large extent of stomach without forming any tumour; which tends to condensation and contraction, rather than to circumferential or interstitial deposit, and in contracting, shrinks into a quasi-cartilaginous mass, but rarely calcifies, and never softens; which only causes ulceration of the mucous membrane very late in the malady; and lastly, which contains scarcely more cell-growth than the cicatrix of a gastric ulcer (p. 241), and is not associated with the presence of cancerous deposits in other organs;[1]—when many (if not all) of these characters are simultaneously present, there need be little hesitation in refusing, to such a lesion, the name of cancer.

Of course, to deny the identity of the malady we are discussing with true gastric cancer by no means requires us to question all relation between the two. It is scarcely possible to doubt that, apart from one lesion sometimes imitating the other, as a result of circumstances incidental to both, there is some close pathological connexion between them; a connexion similar to that which, with equal allowances, may be traced between ulcer and cancer of the stomach. Whether this cirrhotic inflammation is really the analogue of what is termed "fibroid" in other parts of the body it is extremely difficult to conjecture. But it is probable there is no other gastric parallel to this apparently common form of new growth in most other parts of the body. And it is certain that the whole class of non-malignant tumours of the stomach (implying in this term any new growth which is fairly protruded from the serous or mucous coat) quite fails to supply any such deficiency. Doubtless, in these latter tumours, as in cancer of the stomach (compare p. 228), the locality of the adventitious structure has a serious influence on the health and life of the patient; and, through this, on its own size and growth;—so that, for example, there are ample reasons why it should never approach in this organ, the bulk or stage it would acquire in other (*e.g.* sub-cutaneous) tissues. But until we know more of the analogy of these cirrhotic deposits to fibroid, it is useless to discuss that relation to cancer which this analogy would imply; or to propound (what there are strong reasons for conjecturing) the possibility of such deposits, originally non-malignant, being afterwards developed into cancer. Nothing short of careful histories of cases during life, and still more careful examinations of the corresponding specimens after death, in far larger numbers than are at present at our disposal, can afford materials for any safe deductions of this kind.

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[1] A decision sometimes scarcely less difficult than that which concerns the histology of the lesion itself: and respecting which I would suggest that, just as a soft or obsolescent secondary deposit of cancer has often (p. 248) been mistaken for tubercle; so it has perhaps been too readily assumed that hard or enlarged lymphatic glands adjoining a lesion of this kind were cancerous.

The *symptoms* of these cases vary even beyond the wide limits customary in gastric disease. A careful analysis of those examples hitherto recorded suggests the following statements.

In a majority of instances, the lesion is unattended by any symptoms whatever; and is only revealed by examination after a death evidently due to some associated disease. In most of these cases, the deposit is small in amount: regular and even in its disposition relatively to the coats, and to the surface, of the stomach; and (in all probability) slow and gradual of access. Rarely, however, when the lesion is more extensive, it is suggested by what we may term circumstantial symptoms:—for example, an epigastric tumour, formed by the hard contracted stomach, with a duration (10 or 15 years), and at an age (20 to 30), such as are very rare (pp. 215, 223) in cancer of the stomach.

A second and more frequent class offers what may be termed inaugural symptoms. These symptoms are so precisely those of severe gastric disturbance generally—anorexia, local pain and tenderness, vomiting, and febrile reaction—that they need not be described at length: the less so, that they resemble (if not absolutely merge into) those of an allied disease, which we shall presently allude to as suppurative linitis of the stomach. Their sudden access and gradual disappearance, followed (as in some cases) by their relapse or reappearance, and by another subsidence, confirm their import as "inaugural" symptoms; and suggest that they are produced rather by the rapidity of the deposit, and by the local and constitutional reactions thus excited, than by its mere amount (or even extent) in the organ. Such a view is confirmed by the records of some instances of this kind, which have terminated in death a few weeks after the commencement of their symptoms; the necropsy showing a soft albuminous exsudation in large amount.

In the remaining (and more frequent) class of cases, the symptoms, though doubtless often furthered by the immediate influences of the deposit itself, really seem to belong, not so much to the lesion, as to its *sequelæ*; and especially to those local changes already noticed as often resulting from its progress. Many of these *sequelæ* are clearly traceable to a deficiency or failure of the conservative process of muscular hypertrophy. Thus, the contraction of the deposit sometimes produces obstruction of the organ, by constricting the pyloric valve; and this change again, sometimes provokes dilatation of the whole gastric cavity. In like manner, whether the stomach be contracted or dilated, the embarrassment and degeneration of the muscular substance, and the consequent failure of gastric peristalsis, which form the frequent (if not the final) results of the lesion, amount to obstruction (p. 142) scarcely less serious. Bulimia,[1] and suffocative tightness in the epigastrium, are thus often associated with the commoner symptoms of gastric mischief; and vomiting is distinguishable by its excessive frequency, or by the enormous quantity, and fermented or semi-putrid quality, of the substances it expels. Hæmatemesis, too, is sometimes copious and frequent, under circumstances which (as shown by the necropsy) refer it to a mere congestion of the mucous membrane, produced by the deposit itself. Ulceration of the mucous membrane, and inflammation of the peritoneal coat, also bring their own train of symptoms, requiring no special mention. And among other prominent details, I can only recall the comparative rarity of ascites, and the frequency[2] of general dropsy, as having much attracted my notice.

The *diagnosis* of this cirrhotic inflammation depends chiefly on the characteristic features briefly summed up above. Comparing these, for example, with those of cancer, it is obvious that the grounds for identifying during life the malady we are considering, must be sought chiefly in its long duration; in the subordinate rank taken by vomiting and hæmorrhage; in the infrequency and lateness of ulceration; in the absence of secondary cancer; and (last, but not least) in the uniform gastric thickening (rather than tumour) which careful manipulation finds in the epigastrium. To the latter result of personal observation I can only add the presence of great anorexia and intense headache, together with an indistinctness or absence of local pain and tenderness,[1] as sometimes confirming to the conjectures thus adducible from the pathology of the lesion to any individual case.

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[1] Mentioned here from this apparent association, rather than from its presumably having so physical an origin. Many of these cases really involve starvation, which renders bulimia natural enough. (See remarks on DILATATION.)

[2] Suggesting (see note 2 to p. 273) a careful examination of the venous system in necropsies of this kind.

The *treatment*—both dietetic and remedial—of these cases cannot be distinguished, in principle, from that of ulcer or cancer of the stomach; and would differ, in practice, so greatly for each instance, that it is useless to attempt any sketch of it here. Many of the subjects of this malady have been either drunkards or dram-drinkers:—a fact perhaps confirming that analogy to cirrhosis which we have derived exclusively from the anatomy of the disease. How many, it is impossible to say from the records at our disposal. I need hardly recommend, in such instances, the withdrawal of the alcoholic poison, and the substitution of tonics; aided (if necessary) by some unsuspected preparation of opium, in gradually diminished doses. Apart from the remedies demanded by the circumstances or *sequelæ* of the deposit, perhaps the chief indication of treatment may be stated to be—that of favouring (by diet, drugs, and every other means in our power) the nutrition of the patient's organism in general, and the hypertrophy of the muscular coat of his stomach in particular.

SUPPURATION OF THE AREOLAR TISSUE, OR SUPPURATIVE LINITIS.

The disease distinguishable by the above title is more infrequent (though less obscure) than the preceding; to which, however, some of its cases closely approximate. A person, usually from twenty to forty years of age, and previously healthy, is attacked with violent pain and tenderness in the region of the stomach, attended by severe and frequent vomiting, and by high febrile reaction. The pain and vomiting increase in severity, and the tenderness becomes so excessive, as to suggest peritonitis; the more so, that it is often accompanied by some tympanites—which, however, like itself, sometimes differs from that of general peritonitis in being limited to the epigastrium. By and bye, jaundice often comes on. In any case, the febrile excitement rapidly merges into prostration; which is associated with delirium, and ends in death by coma, in from forty-eight hours to a few days from the commencement of the attack.

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[1] Pointing to such irregular phenomena of innervation as the nature and locality of the deposit well explain, or even suggest.

The *necropsy* of such cases shows that the stomach is many (often six or seven) times its normal thickness; and that its rotten, spongy, easily-torn substance is infiltrated with a purulent fluid, which is especially accumulated in its sub-mucous and muscular coats. The latter, indeed, is sometimes completely destroyed; and the former thickened by the effusion into a layer many lines in thickness. The peritoneal and mucous coats are affected in much the same subordinate manner, and variable degree, as in the previous malady. The morbid condition of the former ranges, from a mere dulness or roughness of surface, to a complete (though usually limited) peritonitis; which greatly increases the thickness of the serous membrane; involves the stomach and adjacent viscera; and effuses a substance varying from a solid false membrane, to a sero-purulent fluid, by every conceivable gradation of thickness. That of the latter combines, with the same purulent infiltration and rotten condition which engages the sub-mucous tissue, a marked change in the colour of the free mucous surface of the stomach. This change merges from mere redness, in points or patches of variable size, to an intensely injected or even purplish hue, here and there suggesting (if not amounting to) downright gangrene. In cases of longer standing, all of these appearances are sometimes modified. The mucous membrane either sloughs or ulcerates at various parts of its surface; and the puriform contents of the sub-mucous tissue are thus set free in the interior of the gastric cavity. Hence, when such a stomach is laid open, the mucous membrane is seen to be deficient over patches of variable number and size. These patches often form the orifices of fistulous communications with the suppurating areolar and muscular tissue; so that a probe passed into one of them thence enters a kind of irregular spongy cavity, which sometimes occupies most of the interval between the serous and mucous coats. In other cases, the suppurating areolar and muscular coats are less extensively affected; being occupied by a chain of small scattered abscesses, more or fewer of which have burst through correspondingly small and distant orifices in the mucous membrane. In either case, the determination towards the mucous membrane seems generally to spare the serous membrane any very severe or extensive peritonitis.

The modifications occasionally seen in these appearances suggest that this suppurative malady merges, by no very distinct gradations, into various other diseases. That, in a circumscribed form, it may occasionally inaugurate an ordinary ulcer, perhaps sufficiently appears from the above description. But such a commencement of gastric ulcer is very rare; the depth to which the suppuration extends being generally quite as adverse to its ending by so favourable an event, as is its lateral extension. To peritonitis, its relation is rarely more than that of an imitation, which careful examination generally suffices to dispel. To pyæmia, it is more closely connected. For though, as an element of general pyæmia, its occurrence is (to say the least) extremely infrequent, yet the nature and situation of the lesion claim for it, in some sense, this title. And Dittrich[1] mentions a less copious effusion of this kind as being generally found (in connexion with a similar infiltration of other segments of the alimentary canal) during a fatal epidemic of puerperal fever at Prag. Any more specific relation to phlebitis we cannot at present conjecture.

But it is more interesting to point out the frequency with which this suppurative linitis is allied to the preceding plastic inflammation of the same tissues; as an absolute mixture of the two lesions in varying proportions. Occasionally structures suspiciously like pus-corpuscles may be found sparingly diffused throughout the dense white lymph, or tougher fibres, of the cirrhotic inflammation. Occasionally, according to Dittrich,[2] the margins of the suppurative cavities in the areolar tissue are surrounded by a dense mass of fibroid infiltration, which he regards as either of simultaneous effusion, or of reactive import. But I should conjecture both of these conditions to be less frequent than an admixture I have sometimes seen, in which the periphery of a large solid mass of this fibroid character was soaked in an extensive purulent infiltration of the adjacent areolar tissue. Fully conceding the equivalent character of this admixture, I can hardly frame any other explanation of its occurrence than that suggested by the clinical history of these cases; namely, a suppuration of the edges of a cirrhotic mass, itself deposited in the organ long before.

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[1] Schmidt's "Jahrbuecher," Bd. 72, s. 303.

[2] *Op. cit.*

TUMOURS.

In the stomach, the word "tumour" comprehends a group of diseases far from co-extensive with those associated under the same title in the case of many other parts of the body. For while the stomach perhaps illustrates a general rule in the fact, that it is its connective or areolar tissue which is the usual seat of adventitious deposits or after-growths, it is certain that in this membranous organ (as in the intestine) such growths much more frequently affect the flattened or extended form than, for example, do similar formations in the subcutaneous areolar tissue. Hence a large percentage of those diseases which would otherwise be treated of under the above title, fall under that more natural and accurate arrangement which is implied in grouping them as to their symptoms and appearances, rather than their mere outline or form: the group "tumours" being diminished by the subtraction of a large majority of the cancerous, if not of the fibroid (see p. 278), after-growths.

The very diverse cases included in the phrase "tumours of the stomach" are therefore but a minority of the deposits which produce them. And just as we should find it difficult (if not impossible) to define the precise amount of out-growth or projection from the level of the gastric coats, which would entitle a deposit to such a title; so, on the other hand, the histology of many a pedunculated gastric tumour would refer it to ordinary cancer of the stomach. But, though diverse in nature and histology, the group of after-growths which claim our attention as tumours are practically associated, not only by their outward form, but in some degree by their development and history. Any deposit which is circumscribed and bulky enough, and which projects from the serous or mucous surface of the organ, is of course a swelling or tumour. But the majority of such swellings possess a distinct peduncle. And this fact, which seems to express a tendency of all circumscribed gastric deposits to acquire this form, appears to depend on the original situation of these deposits; which for the most part are not only limited in their lateral extent, but spring from the areolar tissue of the stomach at such a depth, as to belong more or less exclusively to the serous or mucous membrane. Hence as it increases in size, the deposit protrudes before it that gastric lamina which can least resist it: and thus, in anatomical language, "acquires a covering" from the neighbouring serous or mucous membrane. Nothing seems more certain, than that this progress towards either surface, as well as the tendency to pedunculation, are essentially independent of the nature of the tumour itself. Without at all denying that, as regards its subsequent stages and its event, a small villous cancer of the gastric mucous membrane differs from an equally polypoid fatty tumour, just as much as it does in its histology, I would suggest that the clue to a number of the details of the two growths may be found in the above circumstances of the origin and progress common to both. Indeed, the resemblance is rendered doubly important by the fact, that up to a certain date they often have precisely the same symptoms; the gravity of which, by the way, obliges us to interpret the contrast between "malignant" and "benignant" in a very qualified sense as applied to gastric tumour (compare p. 278).

Comparing the external and internal—the peritoneal and the mucous—tumours, it may, I think, be laid down, that the former are not only rarer, but that they are (as a rule) smaller, less distinctly or longly pedunculated, and more frequently fibroid. Sometimes, indeed, their structure so exactly approaches that of an old inflammatory deposit, that it is impossible to avoid referring them to this origin; in spite of their considerable size, and (for example ovoid) shape. Indeed, referring to what has been (p. 138) specified concerning the accretion or drawing out of recent peritoneal lymph; and connecting this with the dull appearance, and the complete agglutination, which are the minimum and maximum relics of peritonitis on this and other parts of the alimentary tube—it is impossible to doubt that an inflammatory exsudation in the peritoneum is subject to even more numerous contingencies than is a similar deposit in the pleura or pericardium. But more frequently, the history of the case, and the histology of the specimen, concur to trace its origin into the sub-serous areolar tissue: into which, for instance, its stalk or peduncle visibly passes; often greatly distorting the subjacent part of the longitudinal (and even the transverse) muscular layer.

The symptoms of such external tumours are extremely uncertain. A small tumour is often unattended by any symptoms whatever; or is only signalized, in rare instances, by obstinate and violent vomiting. A larger tumour, besides the swelling which, if favourably placed, it allows to be detected in the epigastric region, generally gives rise to greater embarrassment of the stomach. And of course its nature, and the rapidity of its growth, often add their characteristics; in the shape of pain, cachexia, adhesive peritonitis, and a variety of other circumstances closely analogous to those already traced in describing the more diffuse cancerous and non-cancerous deposits respectively.

As regards the internal tumours, the fatty generally spring from nearly the centre of the "tunica nervea." The cancerous (which usually have a complexly folded or "villous" surface) are commoner than the simple fibroid. And all are much more disposed to the polypoid form than the preceding; their stalks being not only generally narrower and longer, but appearing to be specifically so, in proportion to the rapidity of their growth and the softness of their tissue, rather than to any other characters their bodies possess.

In respect to symptoms, it need only be said—that these tumours of the gastric cavity produce, on the whole, much more injurious mechanical effects than those which are placed externally: that they disturb digestion, and provoke vomiting, more frequently and seriously; besides at times giving rise to severe pain in the epigastrium, or even to hæmorrhage[1] from

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[1] A hæmorrhage which may be referred to an active (afflux) or passive (obstructive)

the stomach. Of course a pyloric situation is, on every account, the worst they can have: even a small tumour here being capable, not only of bringing about a very serious obstruction (with its *sequelæ* of hypertrophy, dilatation, vomiting, &c.) of the narrow calibre which the stomach possesses at this part; but, as its stalk elongates, of passing into the first part of the duodenum, and superadding a great disturbance of this segment of the intestine. Still, apart from these circumstances (which it is obvious are scarcely more than "separable accidents" of these tumours), they may be present for a considerable time, and may acquire a considerable size, without giving rise to the remotest suspicion of their existence.

HYPERTROPHY.

Hypertrophy of the stomach—a state which we have already described as incident to the muscular coat of this organ in ulcer (p. 140), cancer (p. 251), and cirrhotic inflammation (p. 269); and a term which we have refused (p. 272) to the gastric disease to which it is oftenest applied—only remains to be inquired into as an independent or substantive malady.

To the question—Whether that condition of over-nourishment implied by "hypertrophy" ever constitutes such a malady? —it can only be answered; that our existing information affords no sufficient evidence of anything which could properly be conceived of under this title. It is true that some stomachs (especially, I think, those of old and gluttonous people) are much larger or thicker than others. But, to say nothing of our ignorance of the precise original dimensions of these stomachs, or of the exact range of size and thickness possessed by this and other organs in different individuals (p. 2), size and thickness are so seldom increased simultaneously, and the gastric coats are usually so much softer and more flabby than those of the healthy organ, that it is extremely doubtful whether the condition is one of true hypertrophy or over-

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congestion; and to that mechanical injury to its neighbourhood often brought about by the tumour when it is forcibly acted on by the muscular contractions of the stomach, but of which it would be idle to attempt any stricter classification from the cases hitherto recorded.

nourishment. The organ is larger: because it is either too violently, frequently, or continually stretched, on the one hand; or because it has lost the tone which should enable it to resume and retain its proper dimensions in the intervals of its distension with food, on the other. But it is no more really hypertrophied than the enlarged and pendulous abdomen, to the undue bulk of which it so often contributes.

Nor can any more limited hypertrophy of a single gastric coat be verified by observation. In the mucous membrane there is rarely any appearance of such a process; or, if so, the swelling or thickening which suggest hypertrophy are easily shown by the microscope to be associated with distortion (or even destruction) of its tubes and their contents (p. 268) by a deposit between (and external to) these structures. In the sub-mucous areolar tissue, any apparent overgrowth is also shown by a careful inspection to be a new deposit, associated with injury to both the structure and function (p. 17) of the original tissue. And even in the muscular coat, that true hypertrophy which is so often present as an incident of gastric disease, is always (so far as I am aware) a curative effort of Nature; itself the result of those violent and prolonged efforts by which this coat strives to overcome an obstruction.

The only exceptions to such a rule may perhaps be found in a few cases on record; in which life has been destroyed by a violent and continuous vomiting of several days' or weeks' duration; and the necropsy has subsequently shown nothing unusual in any organ, save a white, hard, contracted state of the pyloric region of the stomach:—a condition which, when closely inspected, has turned out to be independent of all adventitious growth or exsudation.

But while most of these cases strongly suggest a mere contraction of the muscular tunic, without any superadded hypertrophy or increase of its bulk—a passive contraction, such as may well have been the result of that empty state in which the organ was constantly kept during the last few days of life—the slight pyloric thickening verified in the extremely rare remainder seems to be sufficiently explained by the physiology of vomiting, as already described. That the muscular apparatus concerned in this act (p. 52) should in some degree obey the ordinary law of nutrition, and increase in proportion to its use, would be by no means surprising;—the less so, that the pyloric closure generally present in vomiting is in some sense itself an obstruction of the organ. That such an hypertrophy should be so moderate, and so infrequent, as it certainly seems to be, is scarcely more surprising, when we reflect on the prostration which vomiting brings about in all the organs by which it is effected; as well as on the cause—a more or less direct injury of the centre of this co-ordinate nervous act—to which these mysterious and fatal vomitings must undoubtedly be ascribed.

ATROPHY.

Atrophy, the condition antagonist to the preceding, seems to shrink into equally scanty dimensions when examined by the light of experience. Like hypertrophy, it has already (pp. 202, 272), been noticed as an incident in the structural maladies of the stomach: and especially as being produced by interstitial deposits, which, by compressing the original tissues, and thus embarrassing or destroying their vascular and nervous arrangements, cause such a degeneration (and even disappearance) of these tissues, as implies (and indeed expresses) the completest atrophy of their substance. And there are indications of another (and more independent[1]) kind of atrophy which is produced in the unstriped muscular tunic of the stomach by lesions involving its mere disuse. But while, in this subordinate form, atrophy of the stomach is of grave pathological import, as expressing, and inaugurating, changes of the most serious kind, we can scarcely accord it the nosological rank of a substantive or independent malady. Indeed, I think we might go further, and say, that the stomach appears to enjoy something like an immunity from this process; or at any rate that a marked degree of atrophy, chiefly or exclusively involving this organ, is one of the very rarest lesions met with in the human body.

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[1] An independence which is of course better seen in the intestine, as the result of a more distant lesion; but is in exact parallelism with the atrophy of a disused voluntary muscle.

That, in atrophy of the whole organism, the stomach should suffer inclusively—in other words, should decrease in bulk and weight to the extent verified by Bidder and Schmidt[1] for the mucous membranes of the alimentary canal in animals starved to death (about 31 per cent.)—is only what might be expected. But there are appearances of a peculiar resistance to even this inclusive atrophy on the part of the stomach, as contrasted with the intestines. At least I am justified in inferring as much from various instances, both of disease[2] and of chronic starvation,[3] in which the latter segments of the digestive tube have been reduced to an extreme tenuity,[4] in the absence of any corresponding atrophy of the stomach.

Of the records of extreme atrophy of the stomach, almost all exactly correspond with what I have myself observed in less marked instances of the same appearance. A few of them are associated with minor degrees of that softening, already referred to as being chiefly of *post-mortem* occurrence, and as especially marked in childhood. The majority have been found in cases of death by pulmonary consumption, or other disorders generally implying extreme emaciation;—disorders which (you will recollect) have the same general connexion with the greater degrees of gastric solution witnessed in the dead body.

What kind of a malady then do such considerations leave us? Or what more can we say of these cases, than that they are in great degree explained as instances of the stomach sharing in a general process of emaciation and atrophy, which often robs the whole body of two-thirds of its weight, and which is probably[5] capable of reducing the substance of this and other digestive mucous membranes to scarcely less than half their original bulk or quantity. Adding to these facts the contingencies of exfoliation and solution (p. 75) which observation shows to be sometimes mistakeable for increased tenuity, and to be peculiarly frequent and marked in the very class of maladies associated with such marasmus, I confess that all the phenomena of atrophy of the stomach at present known seem to me quite insufficient to establish its claims to rank as a substantive gastric disease.

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[1] "Die Verdauungssafte und der Stoffwechsel," p. 331.

[2] Abercrombie, "Diseases of the Stomach and other Abdominal Viscera," 1830, p. 336.

[3] Donovan, "Dublin Medical Press," 1848, p. 66; also my own observations in cases of gastric ulcer.

[4] In a far less striking degree, such tenuity is illustrated by several instances in which I have distinctly felt the several outlines, and the consistence, of scybala through the walls of both the colon and the belly during life. Most of these were cases of gastric ulceration, and have completely recovered.

[5] Compare Bidder and Schmidt, *loc. cit.*

DILATATION.

It is evident that dilatation of the stomach can only be regarded as morbid, by so much as it exceeds that degree, and duration, of distension, which this organ has necessarily to undergo from the reception of food. And since large variations in this respect are quite compatible with health, while the size of the stomach is itself subject to considerable difference in different individuals (p. 2), it is obvious that we have no precise means of determining what is, or is not, abnormal dilatation. While we have noticed (p. 72) that the appearance itself, as seen in necropsies, is often somewhat equivocal. Hence, for practical purposes, we are obliged to restrict our attention to what are rather the best marked, than the only instances of this condition found in the dead body.

Restricted to these limits, dilatation of the stomach claims our notice under two aspects. On the one hand, it is incidental to various gastric maladies. On the other hand, it is occasionally the symptom (or rather expression) of a mysterious and fatal disease, which is chiefly or exclusively concerned with the stomach. These two varieties may be distinguished as secondary and primary, or casual and idiopathic, dilatation of the stomach respectively. But while such terms are certainly convenient, we must recollect that they are scarcely accurate; since, however diverse in their nature, the conditions they represent merge into each other by numerous gradations of admixture.

The diseases to which dilatation is incident have already been mentioned. In ulcer (p. 141), in cancer (p. 252), and in cirrhotic inflammation (p. 270), we have had occasion to notice, not only the occurrence of dilatation, but also the circumstances by which it is specially furthered and restrained.

Among these we have particularly noticed the state of muscular hypertrophy, as one which is, not so much the antagonist of dilatation, as its natural prophylactic (so to speak) : an effort of nature to stave off dilatation—an effort which, if successful, prevents it altogether; and if not, is yet capable of diminishing its worst effects, and limiting its increase, by being associated with it.

Recalling these incidental dilatations so far as to enumerate the conditions under which they occur, I would suggest the following conclusions :—

1. Dilatation of the stomach is produced by obstruction or occlusion of its calibre. This proposition, which (*mutatis mutandis*) holds good, as you are aware, of the whole intestinal tube, is well illustrated by the frequent results of scirrhus of the pylorus, and of contracted scars of gastric ulcers; two lesions which give rise to what are, on the whole, the most frequent and marked of all gastric dilatations. In minor degrees, such gastric enlargements are very common incidents of both of these maladies. And even the enormous[1] dilatations represented by a gastric capacity of twenty or thirty pints are usually instances of this kind.

2. Dilatation is brought about by the mere destruction of any segment of the gastric muscular coat. Thus the removal of a large ring of this tunic, by an ulceration affecting the circular form, is pretty sure to give rise to a dilatation of the organ behind this loss of continuity. And in some cases it really seems as though a mere embarrassment of peristalsis, by a lesion not destroying any considerable amount of the muscular tissue, produced analogous effects (p. 141).

3. Closely akin to the dilatation brought about by destruction or removal, is that produced by injury, of the muscular coat (and especially by the injury of interstitial deposit), as exemplified in many cases of scirrhus. Here the muscular fibres, compressed by the hard cancerous growth which surrounds their bundles, are distorted in their course, deprived of their supply of blood, and embarrassed in their action, if not

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[1] Even these are probably often understated, for want of the precaution of accurately filling such stomach, at the time of the necropsy, instead of merely emptying and measuring their casual contents.

indeed absolutely incapacitated for all contraction and movement. Under such circumstances, any co-ordinate and effective propulsion is almost as much out of their power as if they were absolutely destroyed. Hence the affected part no longer forwards its contents with the necessary energy and regularity: and their accumulation behind the seat of this diminished or embarrassed peristalsis gives rise to dilatation, differing in little save its degree from that which is produced by the narrowing or occlusion of a similar segment of the stomach.

4. The universal dilatation sometimes seen in cirrhotic inflammation is distinguishable from this last variety in the fact, that it is produced by a more general deposit in the organ; and hence that it engages, not only those parts of the stomach which lie behind the injured muscular fibres, but also those parts which correspond to these fibres themselves. It is possible that this dilatation is sometimes (at least in part) the result of a paralysis (p. 67) of the muscular coat, through nervous tissues which themselves share in the state producing the deposit. But it is more probably connected with the situation, and the consistence, of the inflammatory effusion. And it is certain, both that it is mingled with various grades of hypertrophy, and that it rarely gives rise to such excessive enlargement of the organ as is seen in the above forms of dilatation, which engage the healthy segments of the stomach above (or behind) the lesions by which they are conditionated.

As, in all these incidental forms of dilatation, the enlargement is opposed and restrained by hypertrophy, the conditions which chiefly regulate the latter state have an important influence upon the former. And conversely, while it is of great practical importance to remember that the completeness of curative hypertrophy varies, other things being equal, chiefly with the activity of that nutrition (both local and general) by means of which the over-taxed muscle has to retain its effective force, and to augment its bulk and power; it must not be forgotten that there are marked indications of dilatation itself regulating hypertrophy, by a direct and immediate influence. And it is chiefly by considering the mutual relations of these two states that we gain the clue to their innumerable combinations in different cases.

A careful analysis of the dilatations incident to the three diseases above mentioned (p. 291) tends to show that, in the production of hypertrophy, the rate or rapidity with which the deposit involves the gastric tissues is an important element: so that, for example, the same obstruction which, if of sudden production, brings about great dilatation, will, when of slower development, result in a much smaller degree of dilatation, by permitting a corresponding degree of the antagonist hypertrophy.

It may perhaps be suggested, that this relation admits of a very simple explanation:—that inasmuch as the process of hypertrophy probably implies, not merely an exalted nutrition of the original fibres, but also a formal development of new ones, it may well require a certain time for its production in both striped and unstriped muscle. But it is obvious that this is but a vague and partial way of explaining the fact: and that, assuming its accuracy and completeness, all cases in which the two states concur ought to be hypertrophied in exact proportion to their duration. While we have seen that, not only does no such rule obtain, but that its reversal would more nearly correspond with the facts observed (p. 270).

A more careful consideration seems to show that the influence of rapidity of deposit in opposing hypertrophy is mainly due to the suddenness and amount of the dilatation it brings about. In cirrhotic inflammation it is certainly possible that this extreme and rapid dilatation is sometimes permitted by a morbid condition of the nerves of the stomach—a condition itself capable of opposing hypertrophy by the influence of these nerves on the nutrition of the muscular coat. But as regards that healthier type of dilatation which is exemplified by the obstruction of a scirrhus, or a cicatrix, exclusively involving the pylorus, we may find a more probable explanation in a law of muscular contractility. Just as the sudden application of a heavy weight to the end of a voluntary muscle[1] not only elongates its fibres, but utterly exhausts them of all contractile power; or just as the enormous[2] distension of an occluded intestine soon exhausts and annihilates[1] the writhing contractions by which its muscular coat at first strives to propel its contents past the obstacle; so it seems probable that a rapid dilatation of the stomach may destroy the operancy of its muscular fibres:—not only putting them *"hors de combat"* by stretching; but suspending those nutritive changes which are necessary to their contractility, and are expressed by their contraction. How far each of these effects is "physical" or "vital" it is of little use to inquire. For the antithesis is too clumsy to frame a dilemma; and too inaccurate even to probe the facts, which it would obviously be unable to refute.

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[1] Valentin's "Text-book of Physiology," § 1357. Renshaw, London, 1853.

[2] A similar paralysis seems to occur in the *rumen* of cattle, as the result of a surfeit of rich saccharine grasses, which ferment and distend the organ with gases to a degree said to be often fatal if not relieved by puncture.

But whatever its exact nature, it can scarcely be doubted that the muscular paralysis present in such dilatations of the stomach is closely related to the paralysis which constitutes one of the chief elements of the next class: and that, in so far, it forms the link between the incidental and idiopathic dilatations of the stomach. And for these latter, in spite of their rarity, I must particularly claim your attention; not merely from their pathological interest, but also from their practical bearing upon all the derangements of this important organ.

It occasionally happens, that a patient recovering from fever is suddenly seized with intense pain in the belly; which becomes somewhat tympanitic, and perhaps tender to pressure, though not otherwise suggestive of peritonitis. But even before that brief lapse of time which would often be requisite fully to develope the local symptoms of the latter disease, he either dies of extreme prostration, or, as in most cases (in all I have seen) recovers with extraordinary rapidity.

In alluding to the cause of such intense symptoms, you will hardly suspect me of overlooking a contingency so common as perforation of the ulcerated intestine in typhoid fever; or even as that minor degree of this event (leakage and inflammation of the peritoneal base of typhoid ulcers) which, in the exhaustion consequent on fever, sometimes suffices to cause death, and might elude a slovenly dissector. But though I do not ask you to accept my diagnosis that in the cases I have seen, no perforation was present, or to overlook the circumstance that their successful treatment (by opium, stimulants, and local warmth) happily rendered all verification of this diagnosis impossible; I can state two facts which put such an explanation out of court. (1.) One or two of the cases were unmistakeable examples of the typhus (or non-ulcerous) fever. And (2.) there are authentic records of other cases, in which, after death from this brief disease or paroxysm, a necropsy has shown no structural lesion of any kind, to the most sedulous and skilful examination, save perhaps a pale and relaxed condition of a variable length of intestine.

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[1] The author "On the Physiology of Intestinal Obstruction," *Medical Gazette*, 1849.

In rare instances, after symptoms otherwise closely akin to these have ended in death, the extreme epigastric pain and distension which have pointed to the stomach as the part chiefly affected during life, are confirmed by its inspection after death. The organ is found so enormously dilated as to occupy the greater part of the belly, and conceal the pale and contracted intestines. Its contents are often verified as formed of matters which were taken into it many days before, and which have since undergone a degree of decomposition proportionate to the duration of their sojourn in its cavity. The latter fact obviously proves (p. 63) a derangement, if not a suppression, of the process of secretion proper to the stomach. The mucous membrane, apart from congestion of moderate degree and doubtful import, shows nothing save such an imbibition (of colour, &c.) from the gastric contents, as also seems in part (p. 72) equivocal. And the muscular coat, always of course extremely thinned, is sometimes here and there so stretched, as to suggest a forcible derangement of its layers into a network of scattered fibres—a network which is only filled up into a continuous expanse by the extension, over its interstices, of the other (and especially the areolar) tunics of the organ.

In these acute cases of gastric dilatation, the malady is sometimes clearly traccable to an excess of food: in short, to a surfeit, such as the voracity natural to persons convalescent from fevers and similar disorders (a condition associated with many of these cases) only too often induces. But in other instances, no such cause is present. And in others, somewhat less rapid in their course, but equally obscure in their origin, we may trace a kind of intermediate gradation as respects the important symptom of vomiting; which, usually absent throughout in the above acute instances, and present (with emaciation) throughout the many months' duration of the more chronic ones, ushers in the malady by one or two fits, thereafter remaining absent.

Here then is a class of dilatations, of which these commoner and more typical instances are marked by symptoms, such as permit little doubt, either of their nature in general, or of their diagnosis in particular. Pain, though variable, is usually excessive. Tenderness or soreness is scanty, or even absent. Percussion and auscultation map out the enormously distended stomach (p. 5), and show that it occupies the front of the whole abdominal cavity. The walls of the belly are themselves relaxed, stagnant, and motionless; and (especially in chronic instances where emaciation assists) so thinned as to allow the same statement to be made of the muscular coat of the stomach itself. Vomiting (as you would expect from this condition of the parts by which it would have to be effected[1]) is either reduced to a minimum, or is absent altogether. Lastly, partial or total anorexia, and extreme prostration (as shown by the usual constitutional symptoms of this state) complete the characters of such dilatation; and thus make up an outline, which, on consideration, you will find tolerably excludes the diagnosis of every other disease; as it certainly does that of the other (and more frequent) class of enlargements of the stomach.

The pathology of the malady admits (I think) of but one explanation; to which, obscure and vague as it certainly is, all these marked symptoms converge (as it were) with remarkable unanimity and distinctness. It is evidently a lesion of the nervous tissues connected with the stomach;—a lesion which, whether structural in the sense of being recognisable by the aided or unaided eye, or merely what our ignorance conveniently terms "functional," prostrates (often apparently paralyses) the stomach in all its tissues, and in all their offices. Its mucous and muscular coats, its secretion and motion respectively, are alike interfered with: nay, more, those muscles of the locomotive system which are linked with the contractile tissues of the stomach in the ordinary act of vomiting are also affected. In short, it is not merely the sympathetic, but the cerebro-spinal centre, which is injured or deranged;—a fact which seems to concur with a violent pain and general prostration in pointing to the pre-vertebral centre (or solar plexus) of the former, or to the pneumo-gastric trunks of the latter, as the parts chiefly affected.

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[1] Compare p. 51, *et seq.*

The symptoms which betray the more numerous class of incidental dilatations differ, not only according to the nature of the original malady on which they are engrafted, but according to the amount of the dilatation itself; and, still more, according to the degree of hypertrophy with which it is associated.

But, in most instances, the symptoms developed are simple enough. For it is chiefly in the non-ulcerated cancer, the cicatrized ulcer, and the obscure cirrhotic inflammation, that these dilatations occur: in other words, in lesions of the stomach which are themselves for the time comparatively quiescent and latent.[1]

Enormous dilatation of the organ, as shown by a physical examination of the belly, is of course the first and most important of these symptoms. With it, too, are usually associated others scarcely less characteristic. The vomiting present is so peculiar as almost to be specific of the malady. Coming on every two or three days, and often leaving the patient free from nausea in its intervals, it expels from the enormous stomach, contents the quantity and quality of which conclusively show that they have been gradually accumulating ever since the last attack. The liquid (one, two, or even three gallons) sometimes thus discharged, is distinguished from any ordinary vomit, not only by its quantity, but by its horribly fetid, putrescent odour, its black colour, its spumous yeasty look, and the confervoid growths (generally *sarcinæ,* and often *torulæ cerevisiæ*) which it yields to a microscopic examination. And in spite of this enormous vomiting, the appetite of the patient is generally good; often quite voracious: a condition obviously referable to the same cause as the debility, emaciation, or febrile reaction frequently present—namely, to that starvation which is implied in the failure of the stomach to propel its contents into the duodenum. Indeed, besides this kind of inanition, we may justifiably presume that there is a certain degree of *apepsia,* or failure of the gastric functions: a failure which, perhaps in part inherent to the original malady that provokes the dilatation, or even to the lesion which extreme dilatation may practically be regarded as amounting to, is much more attributable to the decomposition of the food itself. Be this as it may, the unfortunate sufferer, after voiding the contents of the stomach, is irresistibly impelled to recommence filling the organ: and gradually adding one meal to another, at length brings about a state of repletion, and a sense of pain, weight, or distension, which are the preludes to a new attack of vomiting, ending as before. Of course the intervals at which these fits recur are materially influenced by the degree of gastric occlusion, as well as of hypertrophy and dilatation, present; and by the quantity (and even quality) of the food which the patient consumes. And prominent among the symptoms of such a state as the act of vomiting certainly is, perhaps it may still be said to occur with less frequency, and (as it were) with more reluctance, than might be expected from the readiness with which this act seems to accompany other gastric derangements. If this be so, the two classes into which we have divided gastric dilatation meet at more than one point of contact: merging into each other, not only by the analogy already (p. 294) stated; but also by their symptoms. For, just as there are chronic cases in which the gastric paralysis without occlusion which forms the first class, is associated with occasional vomiting; so the frequent (but variable) appearances of gastric paralysis seen in the obstructive dilatation are often associated, towards the close of the disease, with an increased rarity and difficulty of vomiting;—which, indeed, seems to be sometimes suppressed during the last few days of life.

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[1] It is, therefore, not surprising to find there exists the closest resemblance, clinically speaking, between these cases of hypertrophy and dilatation of the stomach, however produced. The mere physiognomy of the patient is to me so characteristic, as to be generally recognised at a glance. And the appearance of the belly, even as seen prior to careful percussion and palpation, is, for the most part, equally characteristic. I cannot attempt verbally to distinguish the thin, sharp, fleshless outlines, and the peculiar colour, which are perhaps the only definable elements of the physiognomy, from other forms of facial emaciation. But I would suggest that it receives a clue (rather than a summary) in the statement that there is a waste of muscular substance in the face disproportionate to the loss of its fatty and subcutaneous textures. In the belly, what is recognised by the eye is perhaps more describable. The wall of the belly, thinned but not softened, sits closely on the subjacent organs; the outlines of which are almost revealed through this covering. Especially is this the case with the stomach, the curves of which are sometimes recognisable with little or no aid from percussion or palpation; and still oftener define, by a slow, powerful peristalsis, the force and situation of which no other segment of the canal can possibly imitate, the nature and even the site of the original gastric lesion.

The preceding remarks on the nature and symptoms of gastric dilatation are such as to dispense with any detailed description of its treatment. For what, indeed, in these distressing cases, can we hope to accomplish? In many instances, absolutely nothing; in others, temporary relief may be given to the patient by a proper system of diet and of stimulants, in small and frequent doses, so as specially to avoid all distension of the organ. Systematic inunction of the belly with olive oil, or (if the patient do not greatly object to its smell) with cod-liver oil, often affords relief, both by its local effect on the muscular wall of the belly, and doubtless by its nutritive absorption. Careful attention to the warmth of the patient's body is also desirable; especially where sedatives are being administered. To this it may be added, that pain indicates opium; that inanition suggests nourishing enemata (see p. 174); and that the distension produced by the decomposition of the food within the stomach is sometimes mitigated by such remedies as the sulphite of soda. But on all these points I may refer to what has been already said in speaking of those diseases by which gastric dilatation is so often caused and accompanied.

SECONDARY INFLAMMATION.

I may end this fragmentary discourse by alluding to a curious pathological condition; hitherto, I believe, unnoticed, save as a casual detail of the isolated records and preparations from which I have collected so much of my information respecting these rarer diseases of the stomach.

In various lesions of the stomach and intestines, death is sometimes immediately preceded (if not brought about) by a violent inflammation of some part of the alimentary tract, at a distance from the original lesion itself. Vivid and continuous redness, often merging, from intense, congestion into a downright blackness suggestive of gangrene, sometimes quite equivalent to the commencement of this state; and copious exsudation, covering the whole of the affected part with a layer of croupy-looking lymph composed of cell-growth (compare p. 87),—are the unmistakeable evidence of inflammation which may be witnessed in such cases. The extent and distribution of the inflammation are subject to considerable variety. Perhaps the duodenum is its commonest situation; but, in different cases, it ranges the whole of the small and large intestines. Sometimes it occupies but an inch or two of the bowel; or engages (it may be) but a part of its periphery. Usually, however, it extends to a length of a foot or more, and surrounds the whole tube. Occasionally there are two, or even three, large patches of such inflammation; separated from each other by a perfectly healthy interval. And lastly, though it seems originally and chiefly to engage the mucous coat, it occasionally involves the whole thickness of the bowel: the muscular and areolar tunics of which become pulpy, brown, and rotten, so that they give way on the slightest handling: while the peritoneum also is injected, or even covered with a film of exsudation.

The secondary relation of this inflammation to the lesions above mentioned is sometimes clearly established by their history: in which, after a long illness referable to the primary lesion, the patient is attacked by all the symptoms of intense enteritis, ending rapidly in death. But the exact nature of the relationship, or the mode in which this sequence is brought about, remains for the present unknown.

The lesions themselves are such as to throw little or no light on this interesting question. Sometimes apparently cancerous, they are in other cases very doubtfully so: in others, evidently cirrhotic or suppurative inflammation of the stomach. Generally (as in these latter cases) the stomach is the seat of the primary lesion. In some cases, however, a tumour or stricture of the rectum or some other segment of bowel forms the original disease. Sometimes, again, it really seems as if an equally discontinuous inflammation was excited in the stomach itself; as a result of old ulceration (compare p. 160), or cirrhotic inflammation, occupying some other parts of its coats.

In other cases, again, it must be owned that the relation of sequence may fairly be called in question; the appearance (and even the history) being not incompatible with the view of a simultaneous involvement of two distant parts of the alimentary canal, by one and the same disease. Now even were such a disease what is usually regarded as a constitutional one, this curious connexion would deserve to be recognised. But it is certainly not to cirrhotic inflammation, or to the ordinary ulcer of the stomach, that the term "constitutional" could be applied in any special sense: still less when we consider that the lesion under discussion (whether secondary or no) rarely offers characters at all approaching those of the other one; but is usually limited to an inflammation of the mucous membrane, neither extending to its submucous tissue on the one hand, nor pushed to the degree of ulceration on the other. Again, it may seem possible that, in regarding the secondary deposit as inflammation, we perhaps overlook cancerous characters which are obscured or hidden by the inflammatory exsudation the adventitious growth has provoked. But any such process would obviously be very unlike that transplantation of cancer-germs which is so frequently exemplified by the hepatic and pulmonary deposits secondary to gastric cancer (p. 245).

On the whole, this association of disease in different parts of the alimentary canal not only offers us a fact worthy of notice, and claiming inquiry, but suggests some interesting conjectures as to its nature and import. And though, among these, the relations of cancer to the other maladies of this tube, and to inflammation in general, as well as to cirrhotic inflammation and ulcer in particular, seem first to claim our notice; yet, even allowing for the difficulties connected with such a statement, I must say that it is not in this direction that the truth seems to be situated. It is to the nervous system that this curious transfer of disease appears perfectly ascribable. It is in the nervous centres—especially the solar plexus—that I should incline to place the seat of this apparently reflex disease; and it is in the peripheric branches which regulate the distribution of blood in the gastric and mesenteric vessels that I should look for the immediate agents or instruments of its production. And while it must be left to Time to confirm or refute this theory, by bringing facts far larger and more accurate than any now at our disposal, I would remind you that, pathologically, it is not unsustained by analogies in other parts of the body, or even in this very canal;[1] and that, practically, it is not unimportant, as showing a new and increased risk, which the development of any serious structural lesion of the alimentary tube may inflict upon other parts of it, hitherto healthy.

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[1] Such as may perhaps be traced in the duodenal ulceration which has been shown by Mr. Curling to be an occasional consequence of large burns of the tegumentary surface.

LECTURE VI.

Dyspepsia.

THOUGH, in discussing the diseases of the stomach, it is impossible to omit all notice of dyspepsia: yet this subject is so wide and complex, that I must disclaim attempting its full description here. The following Lecture will only submit for your consideration some suggestions respecting it; suggestions in which, if you can find anything like a clue to the rational study of this malady—anything which, taken in conjunction with the other topics we have hitherto noticed, and with the physiology of Digestion, may guide you to the principles of the pathology and treatment of indigestion—their purpose will be fully answered.

Dyspepsia—equivalent to difficulty of digestion—is evidently a wide word. One step towards its conversion into a definition we may perhaps take, by pointing out what it evidently implies:—namely, not only that a certain amount of digestion is (though with difficulty) effected, but that this difficulty is not due to any serious structural lesion. To call by the name of "dyspepsia" such an impairment of function as we find associated with cancerous or ulcerous destruction of the gastric tissues, would obviously be almost as absurd as to call strangulation "dyspnœa," or to speak of a beheaded criminal as having been rendered "insensible."

But even limiting the term to this conventional meaning—a difficult character of digestion, unexplained by structural lesion—it may naturally be asked, "How far is dyspepsia a *gastric disease?* In other words, considering that the function of the stomach is but a part (however large or essential) of the total digestive process, how far are disturbances of this process in general to be referred to the stomach?"

In answer to such a question it must be conceded, that not only is disturbance of the gastric function no exclusive cause of dyspepsia, but that it may almost be doubted whether, in the majority of cases of this ailment, the stomach is the organ first and most in fault. Certainly if we contrast the numbers of the affluent and the poorer classes in this country, and the corresponding proportions in which the stomach often has to operate on an excess and deficiency respectively, of the raw materials it elaborates, we may almost infer that, in by far the greater number of cases of dyspepsia which come under the notice of the physician, this organ is perfectly capable of executing any reasonable amount of work.

But such a conclusion by no means disproves the close relation of dyspepsia to derangement of the stomach, or excludes it from consideration among the diseases of this organ. It may be quite true that, in some cases, the stomach is only distressed by the excessive amount of food introduced into it; and is only injured by being overtasked, much as a muscle or a tendon might be. It may be equally true that, in others, it is weakened by want of exercise, or deranged by a surplus of the materials amenable to other parts of the digestive process: that it languishes for want of the protein-compounds it ought to elaborate: or is surcharged by starchy or fatty matters foreign to its office—perhaps often in excess of what can be assimilated by the organs whose function it is to do so. Still since, in a vast majority of cases, the symptoms of dyspepsia are referable mainly to the stomach—since the organ is either primarily or secondarily deranged, and the symptoms of that derangement are the chief phenomena observed by the physician and felt by the patient—we may safely accept the ordinary view, that dyspepsia generally represents a functional malady of the stomach. Doubtless there are many derangements of other parts of the alimentary canal equally entitled to the name of indigestion. But with the exception of intestinal dyspepsias attended by diarrhœa—and often accompanied by such a catarrhal state of the large intestine as amounts to a structural lesion—the symptoms strictly intestinal are generally scanty and obscure, in comparison with those traceable to the stomach. And hence these latter may justifiably be the chief objects of our study. Even were they mere incidents of the malady, so long as we remembered that they were not its essential or sole features, but only those most accessible to our scrutiny, we should be entitled to accord them a notice proportionate with their importance. But when we find (as we do) that the stomach not only always shares, and sometimes almost monopolizes, the derangements called dyspepsia; but that it is the chief source of their most striking symptoms; and that it is also more amenable to treatment than any other part of the digestive canal—we are justified in describing dyspepsia as being, for practical purposes, a gastric disease.

Why the symptoms of dyspepsia refer chiefly to the stomach it is not difficult to understand. The physiology of Digestion affords us plausible grounds for presuming, that the details which distinguish this organ from the remainder of the alimentary canal—especially its shape, its size, its situation, its office, and the peculiarities of innervation associated with them—give it a kind of paramount importance; and render it, in the main, far more sensible to various disturbing agencies, and far more disposed to betray disturbance by abnormal phenomena (such as pain, vomiting, or flatulence) than any other segment of the digestive tube. So that even were purely intestinal dyspepsia much more frequent and important than it seems to be, the study of gastric dyspepsia would still be the best means of approaching its consideration. Apart from the frequent secondary involvement of the stomach, in the intestinal variety, the symptomatology of dyspepsia of both kinds would be best studied from its most distinct and accessible side. In this respect, indeed, the functional derangements of the stomach and intestine do but parallel their structural diseases: in which we often find that lesions, otherwise precisely identical, are betrayed by much more distinct symptoms when located in the stomach than in the bowels; and that, in the latter situation, they are sometimes mistakenly referred to the stomach, owing to those secondary derangements of this organ which they are liable to excite.

Such a view of dyspepsia suggests two considerations as to its diagnosis. Since it represents, not so much a single and substantive disease, as a variety of ailments; and is characterized, not so much by the presence of certain symptoms, as by the absence of structural lesions; the diagnosis of the malady in general, and of any case of it in particular, are opposed by analogous difficulties. As the progress of scientific Medicine has gradually revealed the morbid anatomy of the digestive canal, and thus detected structural disease with increasing accuracy and frequency, the vague (but useful) term "dyspepsia" has acquired a continually more restricted meaning. Nor can we doubt that it is destined to still further limitation; and that, as advancing knowledge brings us better means of investigation, and so enables us to discover and distinguish structural changes of which we now can only observe the functional results, the aggregate of maladies called dyspepsia must undergo successive subtractions, tending more or less completely to its total subdivision into special maladies, and to the removal of this term from our nosology. And while I hope that many of you will hereafter repeat (as you advance in the knowledge of your Art) this increasingly restricted application of the term "dyspepsia," I venture also to predict that, even in the investigation of a single case, you will often find yourselves deducing its dyspeptic nature rather by a gradual exclusion of what it is not, than by any such a sudden recognition of what it is, as, in the substantive diseases of many other organs, often anticipates the result of a systematic inquiry. And (as a kind of corollary to this) let me suggest, that not only does "dyspepsia" still probably include many really structural (if not chronic or permanent) maladies of the organs it affects, but that so closely do its absolute symptoms resemble those of graver diseases, that its importance is enhanced by the possibility of mistaking for it such lesions as cancer or ulcer of the stomach. Sometimes, indeed, such errors are almost unavoidable. But, more frequently, they arise from a forgetfulness of the slight characteristics of these structural diseases in their earliest stages; leading to a hasty conclusion, which a brief suspension of judgment would have prevented, or even reversed. Nor is the stomach the only organ whose lesions may be mistaken or regarded as mere dyspepsia. Just as there is none whose diseases do not react upon the organism generally, and thus influence the nature and amount of food the stomach requires, and the instinctive sensations of hunger or anorexia associated with the fluctuations of these wants,—so it is hardly too much to assert, that every grave constitutional malady has its own dyspepsia; brought about by conditions strictly analogous to those which produce it as an independent ailment; and capable (in those cases in which it is unusually early or prominent) of masking any slight indications of organic mischief in the particular organ to whose lesion it is indirectly due. Much as the progress of physical diagnosis has done for modern Medicine, the warnings of our predecessors as to the danger of mistaking phthisis and other diseases for the dyspepsia secondary to them, are even now worthy of your recollection :—not only because they afford the strongest motive for a thorough inquiry into the condition of all suspected organs in every case of dyspepsia; but also because the physical signs of these diseases are, in rare instances, so indistinct, as to leave us almost exclusively to that keen scrutiny of symptoms, and that mental process of induction, by which the sagacity of the older physicians sometimes arrived at a diagnosis scarcely less accurate than what is now more easily obtained from physical investigations.

The minor degrees of dyspepsia are so common, that few persons in civilized life altogether escape them. Notoriously, most people have at some time or other eaten or drunk things which, in popular phrase, "disagreed" with them. Equally well known is the fact, that the substances thus apt to derange the digestion of one individual are taken with impunity by others; so that, as the proverb states, "one man's meat is another man's poison." And could we suppose (not I am afraid quite an impossibility) that the substance thus productive of derangement was nevertheless so palatable as to be habitually made use of by any person; or that, in the mixture forming his food, the offending article escaped his detection; there can be no doubt that the repeated or continuous symptoms thus produced would at first resemble, and probably at last amount to, an ordinary habitual dyspepsia.

Symptoms of such every-day experience require little description. As you would expect from the nature of the malady, and the functional disturbance to which it seems limited, they usually consist of but moderate degrees of the various phenomena already alluded to as generally characteristic of gastric disease (Lecture I). Thus pain is often limited to weight, tightness, or dull aching: vomiting is rarely more than casual and infrequent, or is reduced to nausea: flatulence is, as a rule, more severe than either: constipation and diarrhœa are comparatively inconstant, and of doubtful import: as is also derangement of appetite. Headache, on the whole, belongs rather to the accidental, than to the habitual, dyspepsias: in other words, is not often intense as well as continuous; but if so, often masks or replaces more local symptoms. General uneasiness, languor, exhaustion, or depression,—by no means unusual associates of habitual dyspepsia,—are attached rather to a concurrence of all the preceding symptoms than to a remarkable prominence of any one or two of them; and are, for obvious reasons, quite as often results of the malady, as elements or constituents of it.

Without pausing, therefore, to trace the rise and progress of an ordinary dyspepsia, as it advances from a casual or accidental indigestion to an habitual and constant malady—without attempting the impossible task of sketching the thousands of forms which a varied degree and number of the preceding symptoms may combine in any given instance to produce—we will assume that the symptoms above enumerated afford us a kind of vague outline of the disease; and proceed to point out one or two of the chief varieties most frequently met with in practice. And you must not be surprised if our varieties, chosen as they are upon what may be called physiognomical grounds, mutually include and decussate with each other; so as to give us what, if we were attempting a true classification, might be logically objected to as a "cross division" of the subject.

First, then, the prominence of any one or two of the above symptoms constitutes a variety of dyspepsia too obvious to require much notice. For example, severe or continuous pain in the epigastric region may be dignified into "cardialgia:" "flatulent dyspepsia," again, is a term equally intelligible: vomiting is so rarely a leading symptom, as to justify a suspicion of some graver malady than dyspepsia, in those cases in which it is disproportionately frequent or severe. Constipation, where really specific to the malady, and not (as it often is) a mere secondary result of the habits of the patient, belongs rather to intestinal than to gastric dyspepsia; a fact in which it resembles the opposite (and less frequent) variety attended by diarrhœa. Both, too, deserve notice, as varieties which often dictate (and amply repay) a treatment specifically directed to these symptoms.

Other varieties of dyspepsia are distinguished by the relation of the symptoms to the period of gastric digestion. The older writers judiciously distinguished what we may term an "ingestive dyspepsia"—an indigestion closely following on the reception of food into the organ,—and gave it, under the term of "morbid sensibility" of the stomach, a prominence to which it is strictly entitled; both as really constituting a severe variety of the malady, and as often including cases of commencing ulceration of the organ. The cases in which dyspeptic symptoms come on more gradually during the activity of the stomach, to end with the propulsion of its contents into the duodenum, are perhaps also entitled to rank as a distinct variety. Those cases in which the access of indigestion concurs with the close of this act are even more numerous and important. In these, again, we may distinguish two sub-varieties: a post-digestive, and a fasting dyspepsia; the first transient, the last enduring until it is relieved by the ingestion of a new meal. Both of these are generally associated with a great disposition to flatulence.

Another classification even more physiological may be also suggested: namely, a division of dyspepsias according to that alimentary constituent, the failure of which to be digested provokes their symptoms.

It will be recollected that, in speaking of the composition of the food in the Lectures on Digestion,[1] its chief ingredients were arranged (in accordance with the classification suggested by Dr. Prout) under the following heads. 1. Albuminous materials or protein-compounds; such as albumen, fibrin, casein, &c. 2. Hydro-carbons or fats; exemplified by butter, oil, animal fats, &c. 3. Hydrates of carbon; a group which includes the various starchy and saccharine substances. 4. Saline constituents which, though less exactly known in respect to their quantity and quality, seem scarcely less important than the preceding. 5. Water; the more or less immediate solvent of some of the above constituents; and the instrument of most of the mechanical and chemical (compare p. 34) phenomena of Life.

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[1] These lectures have been published as an "Introduction to Dietetics." Longmans, 1861.

Now in respect to such a classification, it must be conceded, that in many cases of severe and protracted indigestion, an undue quantity or quality of any one of these constituents often suffices to excite a paroxysm of the disorder. But in others it will be found that only one of these constituents is thus resented: so that, for example, the dyspepsia of one patient is called forth by proteinous substances; that of a second by saccharine or starchy matters; that of a third by oily or fatty articles of food. And even the saline and the aqueous ingredients of food might perhaps be added to this list. At least I have known instances in which persons appeared to be rendered singularly dyspeptic[1] by the saline contents of a hard or calcareous drinking-water; as well as others in which nothing but a strict limitation in the quantity of this diluent (however pure) habitually consumed with their food, sufficed to ward off the malady. Of course similar effects may often be traced to the condiments (for example, the peppers and spices) taken with food. But many of these stimulant and acrid substances really possess noxious characters. And few of them can be fairly regarded as ranking among the physiological constituents of food. Besides, their ill effects are sometimes but secondary and collateral, being referable to the undue quantity of food which they tempt those who use them to consume.

To follow out the above allusions would open up the whole subject of Diet, and so lead us far beyond the limits prescribed for the present course of Lectures. But while I leave it to you to take up the suggestions they offer, I would add that, in the treatment of dyspepsia, this alimentary classification is often of extreme practical value: pointing, as it does, to measures the simplicity and efficacy of which it is difficult to exaggerate. I think, too, that a careful examination would show that each of the several dyspepsias thus distinguished is generally (though not always) associated with certain peculiarities of the symptoms. In the proteinous dyspepsia, for example, of the affluent and sedentary, headache is common: pain is rarely severe; and is often referred to the region behind the sternum, corresponding to the cardiac aperture of the stomach. The starchy dyspepsia[1] of the poorer classes is chiefly distinguished by the extreme flatulence which generally accompanies it. The oily dyspepsia, besides much headache and pain, is usually attended by severe and continuous nausea, or even vomiting. In all these varieties, that excess of the particular constituent which provokes the dyspepsia may of course be either absolute or relative. And under the head of a relative excess, we must probably include, not only a quantity disproportionate to the powers of its special digestive solvent, but also a quantity disproportionate to that of the other alimentary constituents: all of which seem, in their assimilation, to be mutually dependent on each other.

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[1] An effect analogous to the marked injury which such waters often produce on cattle.

Another classification, which is scarcely foreshadowed in the present state of our knowledge, may yet be alluded to, as probably destined hereafter to much greater usefulness in discriminating the functional derangements of the stomach—namely, the grouping of gastric dyspepsias according to the chemical reactions of the fluids expelled from this organ by regurgitation or vomiting. The well-known fact that the fluids thus expelled are in different cases acid, alkaline, and neutral, might suggest corresponding divisions of the malady to which their expulsion is due. But, just as it is obvious that mere acidity or alkalinity would be a very insufficient basis of classification, and might *à priori* (nay more, would as a matter of fact) group together cases in which the same reaction was due to very different constituents; so a little reflection suggests that the physical properties of such fluids (and amongst these properties, their microscopic appearances) are indispensable to the proper confirmation or checking of their more obvious chemical characters. Inquiries of this kind supply some curious details; which, however, would be out of place here, even if I felt justified in anticipating results that require much wider researches on my part. Suffice it to say that acidity, which you might perhaps think generally due to an undue secretion of acid gastric[1] juice, is more frequently the result of such a lactic, acetic, or butyric fermentation of the food as is quite independent of this secretion; that an alkaline reaction is usually derived from saliva, poured forth in abnormal quantity, and swallowed in the intervals of meals—more rarely, and scantily, from bile (p. 15); and lastly, that more neutral fluids appear to be sometimes due simply to the effect of these two opposite reactions in neutralizing each other.

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[1] This form of dyspepsia may be unmistakeably recognised in the sufferings so graphically alluded to by Dr. Livingstone (in his recently published "Travels") as produced by an exclusively starchy food, and cured by meat.

Here is, perhaps, the place to allude to that variety of dyspepsia known under the name of *pyrosis*. Certainly, if one were to adopt the ordinary definition of this malady, I suspect that many of the cases regarded as pyrosis are scarcely referable to it: and especially that its recorded frequency in gastric ulcer[2] is partially attributable to a vague and inaccurate use of this nosological term. And even restricting it to its true meaning —a pain and constriction at the epigastrium, followed by a simple regurgitation of a large quantity of a thin watery fluid—pyrosis must be regarded, not only as an independent malady, but also as an occasional complication of various gastric diseases, both structural and functional.

The more obvious conditions of pyrosis, whether idiopathic or incidental, are evidently of two kinds: the presence of a quantity of this watery fluid on the one hand; and an act of regurgitation on the other.

As regards the first, a careful chemical and microscopical examination generally shows this fluid to consist chiefly of saliva. As such, its expulsion often implies an excessive secretion of this fluid; which is poured forth in excessive quantity as the result either of an improper character of the food,[3] or of a direct gastric irritation;[1] and is conveyed into the stomach, either by unconsciously trickling down the œsophagus, or by being propelled through this tube by a process of deglutition.

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[1] I have, however, reasons for conjecturing that in some of these acid dyspepsias there is a flux or excess of secretion on the part of the stomach: a secretion, not of gastric juice, but of its acid constituent only, or of gastric juice, *minus* the pepsine.

[2] See Lecture III., p. 169.

[3] The true idiopathic pyrosis appears to be common in districts where the inha-

The regurgitation which expels this fluid apparently differs in no degree from the general act of this kind already (p. 50) described. Like it, a shut pylorus, an open cardia, a contracted pyloric sac, and a questionable or scanty abdominal pressure—are the circumstances which presumably bring about the expulsive act. It is doubtful whether the feeling of epigastric constriction which attends this regurgitation is referable to the muscular action of the pyloric end of the stomach, to its distension, or to irritation of the gastric mucous surface. But the former is, for various reasons, the more probable explanation.

How far the mere accumulation of saliva in the stomach is an abnormal act, it is impossible to judge in the present state of our knowledge. In other words, whether the pylorus usually or normally contracts against a moderate quantity of such gastric contents can scarcely be decided. But having verified the occasional presence of bile in the fluid expelled by true pyrosis, I should conjecture, that the contraction of the pyloric valve requisite for great accumulation implies an abnormal quantity or quality of salivary fluid, if not indeed a gastric irritation itself provoking the flux of saliva.

These considerations may dispense with all but an enumeration of the varieties of pyrosis. It undoubtedly occurs in gastric ulcer; and (though I cannot say I have ever verified it myself) in gastric cancer. In like manner, I have seen pyrosis of quite a typical distinctness, in instances where there was every reason to attribute it to the contraction of an ulcerous cicatrix. And lastly, among idiopathic cases, I have known it supply an interesting commentary to the above statements by occurring in the course of a meal (or rather towards its commencement), and yet bringing up pure saliva (from the pyloric sac) quite unmixed

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bitants live on too exclusively vegetable a food; and is said to be specially caused by the association of a sedentary calling with the consumption of large meals of thin, half cooked (and, of course, unmasticated and unsalivated) oatmeal porridge.

[1] In dogs provided with gastric fistulæ, direct irritation of the stomach readily provokes a copious secretion of saliva.

with any of the considerable quantity of food which must then have occupied the cardiac end of the organ.

The *causes* of dyspepsia claim notice, not only from their relation to the scientific aspect of the malady, but from their extreme importance in its treatment. For, since the disease rarely implies any severe or permanent lesion, the mere removal of its cause would often suffice to effect its complete cure. Unhappily, however, it is often no single and simple cause we have to seek out and remove; but so complex an admixture of causes, as almost baffles inquiry, or, even if it permits of successful analysis, defies both physician and patient to remove it.

In an enumeration of the causes of dyspepsia, perhaps undue intellectual exertion claims the first place. The influence of such exertion must not however be measured by its intensity; but rather by its rapidity, its duration, nay, even by the faculties it involves. The constructive mental efforts of genius are eminently wholesome; not because (as is sometimes mistakenly supposed) they are to genius no very severe efforts, but because they demand a concord of faculties, a symmetry of mind, and an application of reason and judgment—in short, a moderate and varied exercise of all the mental powers. Conversely, I think dyspepsia may be caused by a deficiency of mental exertion;—a person accustomed to intellectual toil being rendered amenable to this malady by the loss of labour which habit had made pleasant, and comparatively healthy to him.

Mental anxiety constitutes a cause of dyspepsia which, though allied to the preceding, and often concurrent with it, is a far more efficient agent in producing gastric derangement; and one which sometimes renders the physician as powerless a ministrant to a dyspeptic stomach, as he would be to "a mind diseased" in the Shaksperian sense. How closely all the phenomena of digestion are connected with the mental states is a matter of common experience:—the rice which the Hindoo criminal is unable to moisten with saliva even to avoid detection; the destruction of appetite by bad (or even by good) news; nay, our very forms of speech, which, with little exaggeration, represent a person as "sick" of any person, or thing, or topic —all of these matters of every-day experience conclusively show

how the chemistry of the stomach is subjected to the least material and palpable agents of our life, to that world of thought and emotion which works within every one of us. And indeed there is little need for surprise at this fact. The most zealous morbid anatomist can scarcely doubt that mere variations in the mind sometimes weaken, decay, and even destroy life, without bringing about specific or traceable lesions of organs. And hence it is not surprising that what is true of the aggregate of functions should be true of any one of them; especially of one which, like this, is so closely connected with the general vigour of the frame, and with the special requirements of its every part.

With regard to undue bodily exertion as a cause of dyspepsia, while we may trace similar results to both their excess and deficiency, we must reverse the rule above hinted at in respect to their relative frequency. Among the affluent classes, dyspepsia is much more commonly caused by a deficiency of exercise than by its excess. No doubt the injurious effects of a sedentary life are often aggravated by the brain-work it implies; as well as by that repeated inhalation of impure air which all our ordinary arrangements (as regards house-ventilation) still more or less involve. But its main effect is traceable to a different source;—to that diminution of bodily waste which a want of exercise implies; and especially, to the want of that completeness and perfectness of combustion which muscular exertion ensures to the whole organism. Perhaps, too, we may conjecture another cause for a deterioration of digestion. It seems to me that the waste of the muscular system has a close and essential relation to the amount of the organic principle of gastric digestion; so that, other things being equal, it depends in no small degree on the amount of muscular movement, how much pepsine can be secreted from the blood by the proper gastric tubes. Unfortunately, too, the appetite of the sedentary individual cannot be trusted to maintain that proper adjustment of the *ingesta* to the *egesta* which is its normal office; but often seems rather increased than diminished by a life of confinement:—a fact which may perhaps be explained by the disposition natural to the human mind, to forego moderation in its pleasures, just in proportion as their number is restricted; and to pay all the more attention to the luxury of the table, when circumstances render this the chief or sole enjoyment at command.

On the other hand, however, dyspepsia is also producible by undue muscular exertion. Firstly, as a result of that feverish state which prolonged or excessive muscular efforts bring about; a state which (as many an over-zealous pedestrian can testify) destroys alike the appetite for food, and the capacity of assimilating it. Secondly (as a modification of this law which often merges into it), a similar gastric derangement is producible by a much smaller amount of exertion during the digestive act; in other words, during the period when the blood which ought to be mainly sent to the intestinal canal, to feed those cyclical currents of secretion and absorption going on there, is derived to the muscles, the lungs, and the skin, and thus prevented from effecting the digestive function. From both of these causes combined, I have repeatedly observed grave dyspeptic maladies in persons apparently but little exposed to a malady presumed chiefly to affect the affluent, the sedentary, and the luxurious. I can especially recal some severe dyspepsias of this kind among those indefatigable *galopins* of the public—the Metropolitan letter-carriers.

More effective, however, than all other causes of dyspepsia are those errors of diet to which Physiology and Medicine have long agreed to refer this malady. For few or none of the preceding circumstances would have much influence, if they were but met by suitable adjustments of the quantity and quality of food. The man whose intellectual toil spoils his digestion (and *en revanche* is generally somewhat damaged by the dyspepsia thus brought about) would often have little to complain of were his meals less ample; if his food were less rich, better cooked, and better masticated; his drinks less stimulating; and if both were distributed over a longer period of time, and less carelessly huddled into the stomach. The sedentary, again, might often avoid indigestion by diminishing their food, so as to suit the smaller waste such a mode of life entails upon them. And even prolonged or excessive muscular toil has its proper diet. The hunter or pedestrian who climbs the mountains, or the soldier who makes a forced march, in a state of perpetual fever and perspiration, loathing his food, and unable to digest it, would find (*experto crede*) that if the meat and alcohol foolishly thought necessary to such exertions were replaced by bread, butter, a little fruit, and plenty of cold water, he would feel like a new man. And without pursuing such details any further, we may at least deduce from them, that the diet and regimen of health and disease merge into each other; and enunciate the same principles to which even the cod-liver oil given in phthisis, and the brandy requisite in certain cases and stages of fever, are alike referable;—principles which, while they recall the old adage "every man at forty is either a fool or a physician," suggest that the practitioner who ignored them would vary the alternative (or rather illustrate a third contingency) by supporting both these characters simultaneously.

But in alluding to errors of diet, we must again guard ourselves from taking too unqualified or exclusive a view. Among the affluent classes there can be no doubt that excess of food and drink, either absolutely, or relatively to the peculiar constitution and circumstances of the individual, is a dietetic error by far more common than deficiency. But among the poor, this rule is reversed: and insufficiency of food in general, or of certain of its constituents, is a fruitful source of indigestion;—such as embitters the life, and damages the efficiency, of the children of toil to a scarcely conceivable degree.

It is not always, however, that we can discriminate between excess and deficiency. For example, in the dyspepsia of the poor, we must sometimes doubt whether it is the excess of starchy potatoes, or the deficiency of proteinous meat, and therefore of its proper solvent—gastric juice—which gives rise to the flatulence, and the salivary pyrosis, of the malady. So, on the other hand, the excess of the rich may almost amount to a deficiency: and the habitual ingestion of large quantities of meat may be attended with as habitual a deficiency of vegetable aliment, such as few organizations can want with impunity,[1] save under conditions of life unusually healthy in all other respects. Again the food, however plentiful, may be so ill-chosen, ill-cooked, and ill-masticated, as to defy assimilation; or may be steeped in profuse and complex alcoholic beverages which materially interfere with its solution in the alimentary canal. Or lastly, the excess of some particular alimentary constituent may be only relative; the symptoms it produces being strictly analogous to that variety of dyspepsia which the abuse of cod-liver oil produces:—where, for example, a large dose on an empty stomach produces symptoms similar to those which a pat of butter swallowed under parallel circumstances would bring about; and where the nausea, vomiting, and heartburn thus excited are at once got rid of by dividing the dose, and introducing it into the stomach with (or immediately after) other articles of food.

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[1] An incident illustrative of this fact was mentioned to me on good authority some years ago. A lady, who had been long under the care of a fashionable physician of that day for dyspepsia, was obliged by some novel symptoms to call in a practitioner in the neighbourhood of her country seat. To his plain statement, that she was suffering from scurvy, and required plenty of lemon-juice and vegetables, she rejoined by quoting Dr. Blank's oracular assertion, that a particle of fresh vegetable would kill her; adding that, by his advice, she had long restricted her consumption of vegetable food to a little dry toast. Convinced at last that the

Scarcely to be numbered among errors of diet, and yet inseparably connected with them, is the abuse of alcohol, of tea, of coffee, and of tobacco. The effects of these substances we shall allude to hereafter. At present it may suffice to point out, that the influence of all of them in the production of dyspepsia may be regarded as due to two causes;—namely, to their local, and their general action: to the mode in which they influence the stomach and intestinal canal by coming into direct contact with their mucous surface; and to their influence on nutrition in general, after having been absorbed.

For the sake of convenience, we will eliminate all question as to the deficiency which any excess of these substances indirectly involves; in other words, as to the diminished ingestion of food which the votary of alcohol, tea, or tobacco, often practises. Much as it complicates the result, it is no doubt greatly referable to the constitutional effect of these substances, and can hardly ever be separated from it.

Firstly, then, as regards local effects, if we except that stimulating influence on secretion which a little alcoholic fluid taken before a meal will often excite—the results of all these agents are more or less injurious. Alcohol damages gastric

death by scurvy was, on the whole, the more probable and imminent of the two, she allowed the country practitioner to cure her, both of the disease, and of the preposterous error of diet to which it was due.

digestion, just in proportion to the degree of concentration in which it is added to the gastric juice: and where this degree is great, excites inflammatory phenomena which additionally imply a suspension of all true secretion. Besides, the fermenting substances often present in alcoholic liquids (as in beer, sweet and effervescing wines, or liqueurs) appear to further an analogous (and eminently dyspeptic) fermentative process in the ingesta with which they are mixed. The tannin of tea and coffee probably tends also towards a repressal of secretion.[1] And as regards their other ingredients, it need only be stated, that researches on artificial digestion, and the experience of dyspeptic patients, concur in the propositions:—that water is the best diluent of the digestive fluids; and that, while its goodness is mainly in proportion to its purity, even it may be injurious if taken to excess.

Secondly, as regards the general or constitutional effects of these substances, pray understand clearly that I neither deny nor affirm that there are grounds (moral, social, or what not) for separating and distinguishing their action; and that, in classing them together, I am doing so purely on such physiological grounds as would oblige me, if I were addressing a Chinese audience, to add opium to the list. Tobacco may be the exponent of all the vices; and tea a fertilizing liquid which, applied in proper quantity, encourages the growth of all the virtues. While in respect to the ethics of alcoholic liquids, professors of Teetotal exegesis may explain away those opinions as to the lawfulness and expediency of such drinks which have been handed down to us from the wisest and best of mankind—from St. Paul, and Solomon, and a greater than Solomon—may show that the text of the Evangelist is garbled, and (reversing both the miracle and its motive) turn wine into water to suit their narrow and gloomy views. But we have to consider such questions from a different aspect: and may sum up the chief effect of all these agents in the theory, that, despite great differences of detail, they have much in common. With little convertible material, little substance which the organism can assimilate, they all seem to modify nutrition in much the same way: stimulating the nervous energy of this function; and (in the case of many of them) diminishing the measurable waste of bodily substance—especially reducing the excretions of carbonic acid and urea which chiefly express that waste, to a quantity below the respective amounts which would otherwise be habitually evolved.

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[1] As does probably that infusion of tobacco which the chewing of this narcotic introduces into the stomach.

It must not, however, be supposed that the effects of these different substances are really identical; or even that any one of them acts in exactly the same manner upon a large number of individuals. Nor are we entitled to assume that their effects are really summed up by the two kinds of influence (local and constitutional) above ascribed to them. On the contrary, just as our experience of their effects quite entitles us to believe, that each of them exerts its own specific influences on the nervous and vascular systems; so we may well doubt the complete accuracy of the physico-chemical law just laid down respecting their action. Thus, it seems not improbable that the diminished evolution of carbonic acid referred to is compensated by new varieties of metamorphosis; perhaps even by other products of combustion which at present elude a quantitative[1] (if not qualitative) recognition. At any rate we may doubt whether the whole of this decrease of carbonic acid represents a corresponding economy of waste; and still more, whether, if so, it measures or expresses a corresponding advantage to life and health.

Without digressing any further toward this wide subject of the action of alcohol and its congeners, I may state that experience shows their local effect upon the alimentary canal and upon digestion to be, on the whole, unfavourable: while their general effect of altering (if not disturbing) the balance of nutrition, is often scarcely less detrimental to this function. That they are used by the majority of mankind, and that their moderate enjoyment is quite compatible with health, are facts which by no means countervail this proposition. And that the advantages of their general effects often preponderate over the trifling disturbance they produce locally—that, in short, they are sometimes useful remedies against the very ailments which their abuse (or even their moderate use) can otherwise bring about—is a statement which, while it involves no inherent improbability, rests upon an empirical basis such as defies disproof.

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[1] Among such compensations, it may be specially conjectured that carbonic acid is replaced by an increased oxidation of hydrogen, to form water (p. 61); and urea by ammonia or its carbonate (p. 338).

It may perhaps be useful to end these allusions to the symptoms and causes of dyspepsia, by a brief summary of those conclusions respecting the pathology (or rather the physiology) of the malady, to which they apparently point.

(1.) In spite of the wonderful resistance which the alimentary canal exerts against the substances to whose action it is most exposed, it is so organized, that food which is abnormal in quantity or quality is capable of producing derangement of its functions—in a word, indigestion.

(2.) But abnormal food alone is no sufficient explanation. Apart from those idiosyncrasies which render particular articles of diet hurtful (if not poisonous) to particular individuals, we are forced to assume that dyspepsia in most cases implies a special delicacy or weakness of the organs themselves. For not only do we meet with many instances, in which no amount of dietetic ill-usage perverts their functions; but we observe, that such flaws of the digestive organs can generally be so far prevented and corrected by favourable circumstances of the rest of the body—as that, for example, severe or protracted dyspepsia is rarely found associated with an active life of comparative exposure. It is only thus we can explain the prolonged and severe ill-treatment which these organs often sustain, without any apparent derangement of their functions; as well as the equally well-known fact, that the circumstances best calculated to ensure the health of the organism generally—fresh air, moderate exercise, and activity of mind and body—are singularly efficient as preventives of this malady, and as remedies against it.

The first of these causes of dyspepsia indicates that the liability of the digestive organs to functional derangement is a kind of provision of Nature against graver and more deeply seated disease: a provision such as has considerable analogy to the protective function of pain, and to that mechanism of common sensibility of which it forms a part (p. 44). The position of the alimentary canal (and especially of the stomach) relatively to the food, enables its disturbances to forestall and prevent the mischief that might (and in persons of powerful digestion actually does) gain access to the blood, and, through this fluid, invade other organs. Unchecked, dyspepsia is doubtless not devoid of danger, both by what it thus foreshadows, and by what it can itself produce; just as Physiology must own that—*in* and *per se*—pain is an evil. But it stands (as it were) midway between certain deleterious agents and bad habits on the one hand, and the *penetralia* of the constitution on the other; and, if its warnings are attended to, is often the means of rescuing its victims from the slower and more dangerous consequences of these errors. It is true that no indistinct resemblance of symptoms can entitle us to affirm that dyspepsia often ends in such organic lesions as ulcer or cancer of the stomach. But the clinical history of the latter maladies abundantly justifies a strong suspicion of this kind; a suspicion which therefore suggests the cheering corollary, that the proper treatment of dyspepsia may sometimes ward off the access of these terrible structural diseases. And whatever be the doubt inherent to this suspicion, it is impossible to question the truth, with respect to some other diseases, of the time-honoured *dictum*—that the sufferings of indigestion often call attention to some of those errors of nutrition which, if persisted in, inflict serious or irreparable mischief in the form of tubercle, rheumatism, gout, or calculus: and that they thus increase (rather than diminish) the longevity of those whose prudence does not allow this epigastric monitor to warn them altogether in vain.[1]

TREATMENT.

In treating of the means adapted to the cure of dyspepsia, and allotting to medicines, as distinguished from diet and regimen, priority of notice, I must warn you not to suppose they have any real precedence. In the vast majority of cases, drugs do not cure dyspepsia; do not even effect so much towards a cure as they certainly do in many other maladies.

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[1] On this topic, as well as on the relation of dyspepsia to chronic gastritis, compare the remarks in Lecture II., and the note to p. 215.

They mitigate its symptoms, they diminish its effects; but they hardly touch (if we may draw such a distinction) its nature or essence. And their administration is in no degree a substitute for what (always an important indication of treatment) is in this particular malady the chief object of the physician—the detection and removal of the causes by which it has been brought about. While since, in a large number of instances of dyspepsia, the observance of a proper diet and regimen would ultimately suffice alike to remove all symptoms of the existing malady, and to prevent its return, it may fairly be stated that, for practical purposes, the latter class of remedial agents are by far the more valuable of the two.

If any of you should (not unnaturally) wonder at my bringing into an invidious contrast, measures which it is the duty of the Physician to combine in the treatment of any given case, let me explain that I do so from a motive for which I hope your sympathy. This particular congeries of maladies (the pathology of which we have found is still, from various causes, less precisely and accurately established than that of most other diseases of the alimentary canal) has for centuries been the subject of more quackery than perhaps any other of the ills that flesh is heir to. Not to dwell upon that liability to differences of opinion which the votaries of Medicine proverbially share with those of other Arts and Sciences, and which cannot but be prominent in such a subject as the therapeutical action of drugs—not to trace (what, I think, the most charitable observer of human conduct could scarcely fail to recognise) the germs of quackery in some of the specifics against indigestion which have emanated from the ranks of our own profession, I will only remind you of the systematic impostures which live, and thrive, in great part on the treatment of dyspepsia. As a malady which is often slight, almost always easily curable; which peculiarly affects the affluent and luxurious; and is itself sometimes the result of a form of sensuality which perhaps implies, and certainly produces, a somewhat enfeebled state of the mind—dyspepsia is, from all these reasons, the vantage ground of that numerous (but illegitimate) offspring of Esculapius which is sustained by the trumpet instead of the caduceus. Dyspepsia is, indeed, the malady above all others, in which a variety of quackeries work their pretended cures. It is the efficacy of diet and regimen that explains the small nucleus of facts around which they cluster their mendacious statements. And the very same considerations which thus expose systematic imposture, are those which we must recognise, if we would avoid casual error; if we would know what can be effected by drugs in the treatment of dyspepsia, and which drugs we ought to select. With some doubt as to whether those I recommend to your notice will be found by others to possess all the merits which experience leads me to assign them, and with a full certainty that we have much to learn respecting them, I may at least point out with what cautions they are to be appraised.

A person suffering from dyspepsia takes a certain remedy. He recovers. Therefore he is cured by it. So runs the popular conclusion. But the conscientious physician can accept no such flattering or delusive estimate of his remedy. And if the power of the latter be specifically the subject of inquiry, he asks himself, "Was the recovery due to that spontaneous amendment which sometimes occurs in this disease, as in most other diseases: or was it due to the diet and regimen adopted simultaneously with the drug? Are its traditional virtues, again, in any degree confirmed by its physiological effects on the healthy subject? And, as regards its therapeutical influence, can it be traced apart from diet? Can it be verified as a matter of frequent experience, and (in the case of the stomach) as an immediate result of its ingestion? Lastly, can it be confirmed by the results of interrupting, omitting, and repeating its administration?" Judged of thus strictly, our remedies would doubtless be fewer in number: and discoveries of new specifics less frequent. But the Art of medicine would gain by the extension of its scientific basis. Quackery would lose its chief attractions and excuses, by the exposure of this fundamental delusion of the public as to the cure of disease. The public might become healthier, as well as wiser, by learning that the means of health lie rather in the circumstances with which we constantly surround ourselves, than in rare and exotic drugs. And lastly, our practice would gain far more than an equivalent for a mere number of remedies, in the increased skill and efficiency which would result from concentrating our study on the best way of wielding a few of tried and unquestionable virtues.

Such a scrutiny of our Pharmacopœia would no way affect its value, save to place it, if possible, on a still more irrefragable basis. Nor do those occasional results of the *vis medicatrix Naturæ* (aided perhaps by diet and regimen) which are vaunted as cures by the Homœopath, or which may be deduced from the far rarer (but more trustworthy) observations of our own profession on the natural history of Disease, by any means disprove the value of drugs as the armament of the practical Physician. He has not to answer the abstract question—whether Nature and diet can accomplish the cure of disease, but to discharge the practical duty of ensuring this contingency by every means in his power. And, taking our medicaments at their lowest value, there can be no doubt that their proper use tends to bring about this desirable result more certainly and quickly than would otherwise be the case; sparing suffering; abridging convalescence; and saving the many lives which would succumb before the arrival of less rapid and complete relief. Even in dyspepsia, the palliative effects of drugs are far too precious to be dispensed with. While as regards their less measurable tonic and alterative effects, those who have witnessed their influence among the dyspeptic poor, and against all the terrible concomitants—scanty food, foul air, excessive toil, and intemperance—dyspepsia here possesses, must know that it would be a mockery to restrict this important class of our patients to regimen or *hygiène.* To tell an idle and wealthy dyspeptic that his malady might be cured without drugs, would be a hardy statement, though sometimes a true one. But to recommend dyspeptics in general to dispense with all medicines, would be not only illogical, but absurd: indeed, considering the circumstances of most of our patients, even in the affluent classes, scarcely less foolish or cruel than to withhold a rope from a drowning man, and to advise him to save himself by building a boat.

Counter-irritants are chiefly useful in the severer forms of dyspepsia, attended with great pain. In speaking of their application in ulcer of the stomach (a malady in which they are much more generally important) we have already (p. 166) alluded to details, such as may spare any further discussion of them in connexion with this malady.

The application of heat and cold to the stomach itself, by the introduction of warm liquids and ice respectively, may also be dismissed with slight notice. The former sometimes relieves pain; but requires caution, both as to the temperature adopted, and the selection of cases for its administration. The latter is chiefly useful against vomiting, and (less effectively) against pain. Where any definite effect of this kind is desired, solid ice, in small lumps, or in the semi-solid state of raspings, is preferable to iced liquids.

Pepsine[1] has, I must confess, disappointed me in most of the cases of dyspepsia in which I have tried it; even after a careful selection of those which seemed best adapted for its use. Perhaps it is not often, as already hinted, that dyspepsia is caused by a mere deficiency of gastric juice; and certainly our existing means of diagnosis do not enable us to detect such cases with the accuracy that could be wished. While in many of those varieties of indigestion in which we are entitled to suspect graver and more constitutional causes, it is difficult to see how the scanty solution of a single alimentary constituent (generally taken up in excess of the systemic requirements from even a restricted food) can effect much benefit. Occasionally, indeed, I have found pepsine produce considerable disturbance, even in cases where no great irritability of stomach appeared to be present. I have thought some of these disadvantages might be obviated by using it in the shape of peptone;[2] a form in which I believe it would be of great benefit as a highly nutritive drink or enema in certain œsophageal and gastric lesions (p. 174).

The substances termed *tonics* are often remarkably useful: indeed, unless contra-indicated by great irritability of stomach, may be regarded as almost indispensable. Amongst them the various vegetable bitters claim peculiar notice; not only because, by the common consent of civilized mankind, they stand almost midway between food and medicine, but because their effects (both local and general) in this malady are more

[1] See p. 29.

[2] See p. 33.

uniform and beneficial than those of most other medicines. Some of them (such as calumba) have a special power over nausea and vomiting; most of them increase the appetite; and lastly, their prolonged use produces such invigorating effect on the constitution, as almost suggests some definite chemical purpose being subserved by their addition to the constituents of the organism, beyond any merely alterative effect. Among them may be included one remedy perhaps more frequently useful than any other—namely, quinine; which gives us most of the advantages of bark, where the nauseous and bulky powder, or the decoction, of this efficacious drug could not be borne by the irritable dyspeptic stomach. Gentian, quassia, orange-peel, camomile, and a variety of other bitters, are also at times useful; chiefly, however, either as affording a selection where the idiosyncrasies of patients prevent the use of quinine, or as vehicles and adjuvants for other remedies. As a rule they should be administered, either on an empty stomach, or, if their appetizing effect is desirable, shortly before a meal: but when combined with alkalies, they may often be taken with advantage toward the close of the digestive act.

The more astringent vegetable tonics are especially useful where dyspepsia is attended with any tendency to diarrhœa or to pyrosis. Logwood, kino, catechu, rhatany, may thus be administered: in most cases preferably as the powder or infusion, the alcohol of the tinctures sometimes detracting from their effects.

Various metallic substances ordinarily classed as tonics are also extremely useful in dyspepsia. Especially may we notice the preparations of iron: of which I would say, empirically, that it is in general useful, not only in proportion to the anæmia and general derangement of nutrition present, but in proportion as the habits and circumstances of the patient have prevented his getting that due share of light, air, and exercise, the want of which aggravates (if not causes) so much of the dyspepsia of civilized life. Hence it is more useful in females than in males; more in the sedentary dyspeptic than in the florid over-fed subjects of indigestion whom you will occasionally meet with in country practice. That it has other uses in this malady than those of a mere aliment and tonic can, however, scarcely be doubted: increasing (as it appears[1] to do) both the gastric and intestinal secretions by a specific determination to the mucous structures which furnish them. As regards the cautions required for its use, where there is great nausea and irritability, it must generally be avoided. It should always be given immediately after a meal: and (as a patient is not an ink-bottle) never with (or shortly after) tea. Of its various preparations, the modern citrate is perhaps more readily borne than any, and, in an effervescent form, will sometimes agree with the most delicate stomach. Even of this, however (and *à fortiori* of the more irritant ferruginous salts) I would caution you against giving too large doses, as they have no corresponding advantage, and are liable to provoke nausea and flatulence. The carbonate, phosphate, sulphate, and sesquichloride, form a scale of increasingly irritative preparations: and therefore demand, not only a diminished dose, but an increased dilution. So that, for example, while we may give ten grains of the citrate as a common dose, two grains of the sulphate, and seven or eight minims of the officinal tincture of the sesquichloride, diluted with an ounce of water, are as much as most dyspeptics will safely bear. The oxide of iron, as well as iron filings or raspings, which used formerly to be given in treacle, are now (and I think very properly) almost obsolete. In truth, though these and many other remedies have been indispensable, and even now would be both bearable and valuable to a large proportion of our patients, yet we have no right to risk the inconveniences and accidents which occasionally attend their use, unless some marked superiority can be established for them.

Zinc in the shape of oxide and sulphate is sometimes used: chiefly as an adjuvant to iron, the effects of which it appears to resemble, and to heighten by combination. Bismuth, some of the effects of which may perhaps be regarded as tonic, is still more useful as a remedy against that form of dyspepsia which constitutes the "morbid sensibility of the stomach" specified by the older writers. Here its effects in allaying flatulence and nausea, and in preventing vomiting, and (still more) in checking the pain produced by food, are so marked, that we may fairly accept the term of sedative often applied to it (p. 168).

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[1] Compare p. 55.

The alkalies and acids, as well as various salts formed by their combination, have also been largely used in the treatment of dyspepsia.

The *alkalies* (with which we may conveniently mention their carbonates, as well as the alkaline earths) seem chiefly useful in cases in which the close of the digestive act is attended with much flatulence, regurgitation, and heartburn; where their immediate effects may be attributed to a neutralization of those lactic and acetic acids, which the decomposition of the undigested food can produce. In other cases they appear to bring about general results at least as valuable towards the cure of the malady: preventing, for example, the uric acid sediments associated with some of the more obstinate varieties of the malady; or provoking, it may be, the secretion of the liver, pancreas, or intestines. Both with a view to these local effects, and to avoid the neutralization of the gastric juice, the administration of alkalies should be limited to the latter part of the act of gastric digestion, and to the succeeding period of rest. And in any case, unless some definite constitutional result be sought from their use—when a smaller dose, and a larger dilution, become advisable—these remedies should be regarded only as occasional palliatives, or as temporary measures; and should not be pursued for a longer period than a few weeks at a time.

Acids are regarded chiefly as tonics; with local effects which, whatever their minor differences, probably agree in furthering gastric secretion, as well as in aiding the solvent powers of the juice already poured out by the stomach. The differences of their constitutional effects have yet to be established on physicochemical grounds: but the astringent effects long ascribed to sulphuric acid promise to constitute a distinction. They are all contra-indicated by great irritability of stomach. It need hardly be said that such potent remedies should only be given in a small and dilute dose (♏v—xx of the dilute acids, P. L.), and that care should be taken to protect the teeth of the patient from their corrosive effects. They should be administered during or immediately after a meal; and, so far as I can judge, are more efficacious, and less likely to disagree, when mixed with pure or distilled water than in any other combination:[1] a point (by the way) in which they differ from alkaline carbonates, which may often be combined with bitters, not only with no loss of effect, but even with an increase of power. Lastly, it is said that acids and alkalies are sometimes of great service when given alternately: the former just before or during a meal, and the latter an hour or two after: a mode of administration which is certainly of use in some forms of chronic constitutional and visceral disease, but which savours of a polypharmacy, such as is rarely necessary in this malady, and should always if possible be avoided.

Of those various *salts* of the alkalies and alkaline earths which are presumed to have effects distinct from their aperient action, we need only select two for special mention—the iodide of potassium, and the hyposulphite of soda. The latter appears to substantiate the claims made for it by Dr. Jenner a few years ago, as a remedy against flatulent dyspepsia: its efficacy seeming due to its powers of checking decomposition in the food, as well as to its local effects on the stomach itself. It may be given in the same doses and combinations, and with the same limitations, as the alkaline carbonates, the effects of which it somewhat resembles. The iodide of potassium I am in the habit of prescribing chiefly in those cases of flatulent dyspepsia in which—whether from a too starchy diet, deficient or hasty mastication, decayed teeth, the abuse of tobacco, or other causes—the salivary secretion seems either deficient in quantity, or faulty (for example, acid) in quality. In such cases, a very small dose[2] will suffice: from one to two grains in combination with seven or ten of bicarbonate of potassa, sometimes effecting a marked change after two or three administrations.

Aperients constitute a means of treatment the proper appreciation of which is of the first importance. When a person over-eats and drinks himself into what used to be called a surfeit, but is now more politely (but less accurately) named an indigestion, he naturally thinks of removing the cause of the mischief, if Nature have not already done so by a spontaneous vomiting or diarrhœa. The old Roman epicures adopted the quickest and most effective means, an emetic: which could be speedily followed by another feast. The modern votary of good living clears off the effect of his excesses by a purgative; a means of relief in which, with less to excite disgust, and perhaps more to relieve the system, the same principle may still be traced.

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[1] Even quinine is scarcely an exception to this rule, when the effects of the acid are desired.

[2] I could bring forward several instances of the facility with which downright iodism has resulted from small doses (*e.g.* three grains thrice repeated, and even less than one grain), in young and robust persons of both sexes. What graver accidents (delirium and even mania) may follow its abuse, I only know by hearsay, as having been observed by some of the leading living members of the profession.

But a physician is not a *censor morum:* and if I call attention to this method of using purgatives in the treatment of dyspepsia, it is only with the object of pointing out its mischievous effects in a medical point of view. It cannot be doubted that in these effects of intemperance, as well as in many casual (and even habitual) dyspepsias which are quite independent of all such excess, many of the most distressing symptoms may be suddenly and greatly relieved by a smart purgative: and especially by some such purgative as the well-known blue pill and black draught. But nothing can render this immediate relief permanent save the adoption of that diet and regimen, and those general remedies, which form the staple treatment of dyspepsia. I would go further, and say that in many instances the immediate relief thus obtained is very evanescent: and, without any concurrent indiscretion, leaves the patient in a few days no better than before, as regards the local symptoms of his malady; but considerably worse as regards that general debility and *malaise* which constitute its effects, and which purgatives (and especially mercurial purgatives) are themselves calculated to produce. And of the habitual or even repeated use of such measures, I can only express an unqualified condemnation.

Indeed, with respect to the use of aperients in dyspepsia generally, perhaps the following rules may be laid down. They are of great service in emergencies: of little service (almost of disservice) as prominent features in the course of treatment; which ought almost always to aim at such a tonic effect as is incompatible with habitual purgation. Against habitual constipation, they are generally ineffective: and can in most cases be replaced (or substituted) by proper attention to diet. In administering them, the object should be, to accomplish the desired effect with the smallest possible doses. This indication may be greatly aided by giving them on an empty stomach: either before dinner, for example, in the case of a slowly acting pill, or early in the morning in that of a more rapidly acting liquid. As a rule, the pill[1] form is preferable, especially where the aperient is often repeated; the liquid aperients (especially the saline) often acting injuriously on an irritable stomach. Castor oil is, in many constitutions, an exception to this rule: but then, again, it is sometimes difficult to predict the exact effect of a given quantity: in other words, to hit the minimum dose which is all we can give without preparing for that reactive constipation generally observable after an artificial (as well as an idiopathic) diarrhœa.

Sedatives are of course often indicated in a malady so painful as dyspepsia. And amongst them, opium, in its various forms, retains the pre-eminent value it possesses in other diseases. As you would expect, these remedies are useful chiefly as palliatives; and especially as anodynes in the more painful forms of indigestion. In the morbid irritability already alluded to, their use is almost indispensable. But in the purer or simpler forms of the malady, it is our object, on constitutional grounds, to administer them as rarely as possible; at any rate to avoid an habitual dependence on their use, which shares some of the nutritional disadvantages ascribable to alcohol and other stimuli. The time, dose, and mode of administering them, are matters for decision in each case: and are regulated chiefly by the amount and duration of effect desired. Thus, to relieve a sudden attack, we should give the drug in solution: to prevent or alleviate severe habitual pain after meals, and in an irritable stomach, a pill of morphia or extract of opium, at such a time before the attack as allows the effect of the drug to anticipate the pain. In such attacks, however, few forms of sedative are more generally useful than the compound kino powder in combination with bismuth (compare p. 169).

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[1] In many instances this rule will also be found applicable to tonics (compare p. 178).

Time fails me to sift the claims of a variety of other remedies; mercury, silver, arsenic, prussic acid, strychnia, and some other vegetable alkaloids. Concerning mercury I would say that, while it is quite possible that maladies in which dyspepsia is prominent may sometimes require its administration, its habitual use, even as a purgative, seems to me not without detriment; and that its more marked constitutional effects are generally most mischievous. And when I have told you that prussic acid will often allay vomiting, and that strychnia is alleged to be an excellent remedy against habitual costiveness, I have said all that is suggested to me by the mention of these drugs. If you think this a meagre account of such celebrated remedies, pray remember that I am speaking mainly from experience, which is, in their case, to a great extent negative. After nearly twenty years of almost uninterrupted attendance on the sick, fifteen of which have been spent in metropolitan Hospitals or Dispensaries, where the successful treatment of dyspepsia has been one of my most common professional duties, I must confess that I rarely use the above energetic drugs, simply because I rarely require them. And without asking you to follow my example, or to overlook the strong testimony in their favour of other observers at least as well qualified to form an opinion, I will end by quoting to you a passage from an admirable work on this subject by one of the first physicians in Europe, who has lately died, full of years, and of honours; and leave it to you to judge whether these remedies may not be really contra-indicated, even though occasionally useful:—whether, in short, it may not be a question of tact and prudence, and conscientious care for the public, rather than of abstract therapeutical operancy, which we have practically to decide respecting them.

"I have a great disinclination, I confess, for such remedies, which bring with them so great and incontestable perils, and the good effects of which are, in my opinion, very uncertain. These perils are capable of being prevented, it is true, if the physician is always attentive to everything he prescribes, if he carefully reads over his prescription; if this is written in such a way that there can be no possible uncertainty as to its words and figures; if the druggist and his assistants are protected from all distraction, and all error; if the remedy is given to the patient exactly at the intervals, and in the doses, indicated; if, (unlike what has often happened) there is no giving in a single day the accumulated doses of those which have preceded it; if, after one parcel of strychnia is exhausted, that which follows is neither purer nor stronger. But how can we be sure that all these conditions will be observed, *each* and *every day* on the part of *every one* who has to fulfil them? If, in spite of these dangers, strychnia really cured dyspepsias against which every other remedy had failed, I would myself resort to it. But when I look back to the past, when I see dyspepsia almost constantly due to the errors of hygiène which maintain it so long as they are not removed, when it must be evident to every one that no medicament, strychnia or anything else, can be a substitute for the removal of these causes, and when, in the few cases where I have tried it, I have not obtained any manifestly favourable effect, I do not know how I can recommend its administration."[1]

Diet we shall not dwell upon. For the physiology of digestion has already taught you principles by which to judge of the proper quantity, quality, and frequency of the meals which a healthy person (and *à fortiori,* a dyspeptic) ought to take; as well as the importance of proper cookery, and of that mastication and insalivation which is still more indispensable as a preparation of the food for the digestive process. To inquire in what way the practice of your patient deviates from these physiological principles, and what idiosyncrasies heighten or modify their application to his case, and to prescribe more natural and wholesome rules of alimentation, is of course your chief duty as regards his diet. And there are few rules which could be briefly laid down, so as to guide you in selecting, from the numberless modifications of the diet of health, those which it is from time to time advisable to adopt. Hence the following allusions may suffice.

In respect to *quantity* of food, of course we have often to reduce it in the first instance far below what it would be necessary or proper to recommend for a permanency. In many cases of dyspepsia, whatever is superfluous is hurtful; and therefore we have first to effect the partial restoration of the digestive process by a diet adapted to its feeble powers, and then gradually to revert to a more liberal scale of food. Where the stomach itself is chiefly or primarily in fault, we must just as obviously apply this principle to those protein-compounds which it is its special office to dissolve: reducing their quantity; securing their proper dilution and mixture with other alimentary principles: and especially, giving them in such small and frequent doses, or even in such a bland and pulpy consistence, as will allow them to pass rapidly out of the organ after being impregnated with its secretion of gastric juice, so as to receive their solution in the intestine (pp. 184, 185).

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[1] Chomel, "Des Dyspepsies." Paris, 1857, p. 228.

As to the use of *alcohol* in dyspepsia, while freely conceding that every case is a rule to itself, and must never be decided on general principles, I would offer the following hints. First of all, let me beg of you on no account to endanger the welfare of a reformed drunkard who has been rescued by Teetotalism from death, by prescribing alcoholic drinks in any form whatever. I have known one or two awful relapses, and inconceivable misery (both to the patient and his family), produced by carelessness of this kind. It is much better that you should candidly inform such an one, that his laudable self-denial requires certain precautions; that it demands parallel temperance in his eating; and that just as, morally, it may be doubted whether he is entitled to substitute intemperance of thought and speech for intemperance of act, or to replace the privacy and penitence which befit a repentant debauchee by the public vituperation of what others have made a lawful and innocent indulgence—so, medically, he must be looked upon as merely substituting one error for another, if, from drinking to excess, he takes to eating too large or luxurious a food. Indeed, supposing the Teetotaller's avocations do not demand excessive and continuous toil, he digests so much more completely (at least, such is my opinion) what he does take, that he requires less (rather than more) food than a person in affluent circumstances who moderately indulges in alcohol. While he certainly has lost that safeguard which alcohol in some degree affords against the immediate results of over-eating.

And conversely, the uses of alcohol—which merge, I confess, by no very marked line into its abuses—may also be traced in a few words continuing the above statements. As regards the organism generally, alcohol not only enables a man, by its general effects, to sustain health on food otherwise insufficient, and against toil otherwise injurious, but it also obviates—perhaps oftener defers and accumulates—some of the direct consequences of *gourmandise.* That good eating requires good drinking, has in all ages of the world been admitted: and to select the most invidious illustrations of this fact, the gigantic meals which are sometimes ended by the slow soaking in dilute alcohol of almost all the food taken—so as to check alike digestion and decomposition, and enable the canal to void its contents after a very scanty absorption of their nutritious principles into the blood-vessels—would scarcely be compatible with the health sometimes associated with them, save by an effect of this kind.

The benefits of alcohol in dyspepsia are these. Partly by its local, more perhaps by its general effects, it sometimes gives an appetite to a person otherwise incapable of taking food. Many persons, for example, half dyspeptic, half exhausted, require some such preparation or accompaniment for the chief meal of the day. Similar influences are traced in the increased secretion which its stimulating effects often seem to bring about; an increase well shewn in the singular manner in which those who only take these fluids occasionally find that a small quantity of wine has to be substituted by a large quantity of water. Lastly, there are cases (and these not infrequent) in which a suitable alcoholic fluid seems to facilitate the whole digestive process, and reverses what I believe to be the rule, even for dyspeptics—namely, that if the food be rightly chosen and prepared, digestion is much better conducted altogether without alcoholic liquids. That they render digestion slower, and more precarious, and increase the feverishness from which dyspeptics often suffer after meals, is undoubted. And I have made some observations which justify me in suspecting that, even apart from any sensible derangement of digestion, the moderate use of alcohol largely increases the ammoniacal constituent of the fæces: a result which, if confirmed, would indicate that the diminution of daily urea under its use may have some substitute in other channels of excretion (compare p. 321).

As to the variety of alcoholic fluid to be used, much of course depends on the peculiarities of the case. But from reasons already alluded to, the purer and more dilute the alcoholic mixture, the less danger is there of its acting injuriously on the digestive process. Hence a small quantity of brandy, or twice the quantity of dry sherry, diluted with pure cold water, is generally preferable, both to beer, and to the sweet and factitious ports and sherries consumed in this country. Champagne and the other effervescing wines are mostly injurious. The purer and more natural wines of French and German growth, either diluted or undiluted, are admissible in a vast number of cases in which the preceding are hurtful. And lastly, where an alcoholic beverage is indicated by the general state of the organism, but deranges digestion, the expedient of its larger dilution, or (if this be unsuccessful) of its complete disseverance from the food (or meal-time), will sometimes obviate all such derangement of this process.

Regimen.—There are many other measures, such as we include under the term "regimen," which are of extreme value in the more protracted and obstinate cases of this distressing malady. The importance of fresh air, of exercise, of the proper regulation of the functions of the skin, of the observance of a period of rest after meals, of the repose of an habitually overtaxed brain, and various circumstances of like nature—claim attention under this head. So, also, much might be said of such remedies as foreign travel, mineral waters, and the cold-water treatment. Mineral waters as therapeutical agents can hardly be valued too highly. It is certain that many of them are sustained, without inconvenience, by stomachs far too irritable to support the artificial mixtures prepared by the druggist in accordance with our prescriptions; and equally certain that they introduce into the system considerable quantities of the very remedies (chalybeates, salines, and aperients) which we are compelled to resort to as remedies in many varieties of dyspepsia. And whatever their "alterative" powers, the circumstances which attend their administration are scarcely less beneficial; and especially, permit the enforcement of rules of life, which we sometimes despair of our patients following, so long as they pursue their ordinary avocations, and mix in their customary society. The effects of pure water (as applied externally and internally by the Hydropaths) in dyspepsia have yet to be ascertained. Judging from the evidence at present accessible, it is sometimes of great benefit. But I need hardly say that, when it is applied by persons who make it a system, and regard it as a cure for all diseases (at any rate as a substitute for most other means of treatment), we cannot expect that selection of suitable cases, which can render its results either safe to the patient, or conclusive to the Profession. And from analogy[1] and experience we may safely assert, that most (if not all) of the benefits ascribed to it are equally explained by the fresh air, mental quiet, early rising, simple food, and copious exercise, which the subjects of the Hydropathic course adopt, in conjunction with packings and douches:—perhaps after having steadily disobeyed every such recommendation on the part of their more legitimate medical advisers for months and years before.

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[1] The speedy and marked benefits derivable from but a part of such a regimen are well exemplified in what is termed the "training" of prize-fighters; and in the pedestrian tours from which, annually, thousands of exhausted citizens date a new lease of health.

LECTURE VII.

Gastric Phthisis.

The course of pulmonary phthisis is usually accompanied by disturbances of digestion which, even when they remain strictly subordinate to the other symptoms, suffice to show that the stomach is indirectly involved in the malady. Evidence of such indigestion is seen in the capricious defective appetite; the eructations; the regurgitations of food or of acrid sour liquids; the vomiting; and the various degrees and kinds of pain after food, from heartburn to severe pain and soreness at the epigastrium;—symptoms which are rarely absent, in some form or other, from the greater part of the visible duration of the thoracic disease.

To trace, however, this dyspepsia to its doubtless diverse, and often complex causes, much more to refer its several elementary phenomena to their respective sources, is utterly impossible. Even to enumerate its leading varieties is by no means easy. In some instances, it is fair to infer that the dyspepsia causes the phthisis; which, in point of time, it certainly precedes and ushers in. In other cases, perhaps more numerous, the thoracic lesion and the gastric disturbance seem to be twin effects of a common cause—a bad or cachectic state of the constitution. Far oftener, I believe, the dyspepsia, as well as the various appearances which we sum up by the word cachexia (so far as the word has any exact meaning), is itself the result of the injurious reaction of tubercular deposits—dying, dead, or decomposing—in the tissues of the body or the system at large. Such dyspepsia merges, with no very distinct line of demarcation, into the large and obvious class of indigestions in which the pulmonic lesions set up an irritative fever, having a dyspeptic element evidently analogous to the digestive disturbance attending an ordinary fracture or wound. At a later period of the pulmonary destruction, there are not wanting indications that undue bodily waste, sweating, suppuration, and the toxæmia which the partial putrefaction of various pulmonic tissues produces in the adjacent blood, all exert an influence more or less characteristic on nutrition, and therefore on digestion. And here, again, we approach a more specifically pulmonic causation of these dyspepsias :—(1) insufficient aëration of blood, and the results (many of them it may be suspected less of degree than of kind) which imperfect oxidation of the blood brings about in the chemistry of the organism generally; and which we indicate (rather than sum up) by such words as incomplete combustion, defective elimination, and the like ;—(2) nervous irritation and exhaustion, not only such as are producible by lesions of all parts of the body, but such as specially oppress a function which, unlike that of many other structures, must continue uninterruptedly in order that life may be carried on, and which can only be effected (as the physiology of respiration shows) by an expenditure of energy continually increased[1] in proportion as the diseased organ, itself becoming more defective, continually deteriorates in respect of even the simplest physical conditions[2] of its action. To a similar nervous origin we may refer the vomiting which, in many cases of phthisis, is scarcely to be considered in its ordinary light as a symptom of dyspepsia. The gush of emesis which ends a fit of cough in advanced consumption seems strictly comparable to the vomiting which occurs in advanced whooping-cough ; and indeed, is analogous to the retchings which, in bilious or intemperate persons, are provoked by the gargling of the mouth and brushing of the teeth incidental to their morning ablutions. For in all of these vomitings, an enfeebled nervous apparatus permits or conditionates a kind of morbid co-ordination between its various parts ; which lose, as it were, their due independence, and become liable to be drawn into a simultaneous action ; an extension or transference of their natural working in some sense analogous to that abnormal protraction and extension of muscular action which constitutes an ordinary cramp.

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[1] Of such an increased expenditure of energy, the increased frequency of breathing constitutes in the main a tolerable exponent ; especially as the diminished efficiency of breathing, by affecting unequally various parts of the apparatus, increases disproportionately the labour or energy claimed from the least diseased portions.

[2] Conditions, perhaps, easiest followed out into their details under the physiological grouping of the chest, the lung, and the air-tubes ; and the shares respectively taken by these structures in the act of breathing.

With such diversities of dyspepsia incident to various stages of the course of pulmonary phthisis, it need hardly be said that the collective gastric disturbance of any particular case often defies an exact pathological analysis. But the importance of recognising these diverse forms or causes of dyspepsia by no means rests on the facility or usefulness of appraising the shares which they respectively take in the casuistry of the disease. It is rather as a clue to the gastric phenomena of phthisis in general, and as an explanation of the manifold degrees and kinds of indigestion met with in this malady, that an enumeration like the above finds its justification.

It has the further advantage, too, of forming, by way of exclusion, a group of cases which, on clinical and practical grounds, deserves especial study; and which I propose to consider as *gastric phthisis.*

Without denying that in many cases the dyspepsias incidental to pulmonary consumption merge into "gastric phthisis" by almost imperceptible gradations of symptoms, I can confidently affirm that in the study of gastric disease, increasing experience continually brings this condition before me with increasing prominence; and that, while the latency of the thoracic mischief, and the prominence of the gastric symptoms, would alone suffice to justify their distinction; the cases themselves, whatever may be the diversity of their nature as regards histological details, possess sufficient uniformity in the outline of their symptoms, and in the plan of their successful treatment, to fall naturally within a common description.

The outline of a typical case of this kind may be drawn somewhat as follows. The patient, usually under thirty-five years of age, feels the first approach of the malady as an "indigestion;" an epithet which, on inquiry, resolves itself into a pain beginning between the first and second hours after food, and going off gradually. At first such a pain often follows but one of the daily meals; perhaps oftenest a full morning repast. It rarely brings with it any flatulence; and is still more rarely relieved by eructation. As the malady advances, the pain becomes more frequent, and follows all the meals; only distinguishing, by attacks of unusual severity, those in which the food is more copious in quantity, or more solid and indigestible in quality. By and by, the sickening depression which has gradually been recognised as an element of the increasing pain diverges into distinct nausea; and this again soon provokes retching; which, in its turn, sometimes gradually deepens into vomiting. Often, however, the latter symptom remains long or permanently absent. If present, it is only rarely, or after long persistence, that it brings back from the stomach any of its alimentary contents; and even then scarcely ever unloads the organ, much less relieves the pain by which it is preceded. By longer continuance, the pain and retching become more severe, and more easily provoked; and therefore continually approach the period of taking food, so as not only to follow it by a shorter interval, but at length to limit the meal to little more than painful and unavailing attempts to take food, the suffering which immediately follows its deglutition becoming almost unbearable. The climax of gastric disturbance thus attained is, in rare instances, itself the chief cause and immediate forerunner of death. But it much more commonly either inaugurates a rapid infiltration of the lungs with tuberculous deposit, or is displaced by the thoracic symptoms of tubercle already deposited; to alternate (it may be) with such symptoms during the brief remainder of life. In other cases, the dyspepsia amends spontaneously, or is vanquished by appropriate treatment; and the patient, slowly recovering flesh and strength, advances towards that imperfect health which, in so many instances, is associated with the retardation or arrest of the progress of tuberculosis; perhaps until the infirmities of declining years, mingled with the insidious symptoms of the malady, leave us in doubt to which of these two causes—natural decay or tubercular disease—we must chiefly refer the eventual death.

A closer scrutiny of these cases from their clinical side—in other words, with the very natural object of making their detection and cure paramount to any more systematic or scientific study—leads us first to the question of their connexion with other forms of phthisis among the near relatives of the patient.

In respect to this important point, experience seems to show that the ordinary rule as to the frequency[1] of such a connexion holds good: qualified, rather than really subverted, by the following exceptions. First, the rule applies to a somewhat smaller percentage of these cases of gastric phthisis than of ordinary pulmonary tubercle: in other words, whatever slender aid to diagnosis the history of phthisis in the family can afford is oftener absent. Secondly, there is a manifest tendency to the affection of various members of the same family with a similar or analogous form of phthisis: at least in so far as that we may occasionally find the gastric phthisis—itself rare—occurring in two or more such relatives; and much more frequently may observe its occurrence in one relative, while another succumbs to the characteristic intestinal form of tuberculosis. Of these two qualifications, the former is the more striking and important: certainly, my own experience enables me to recal quite a disproportionate number of cases in which the gastric form of phthisis has attacked persons whose family history was free from all suspicion of tubercle; and whose active, open-air habits were, to all appearance, very unlikely to invite it.

The diagnostic element afforded by an examination of the chest in these cases is one which, practically, may be difficult to detect, but logically, is susceptible of an exact definition. Paradoxical as it may sound to say so, it is quite as much by the absence of some thoracic signs, as by the presence of others, that the results of careful auscultation indicate a given case to belong to the category of gastric phthisis. Large or advanced lesions of the lungs, just as they are not likely to be overlooked on the one hand, so, on the other, they suggest any concurrent gastric disturbance to be of that kind of which we have already enumerated several forms; all being in great degree the frequent and necessary (though indirect) results of the lesions themselves. But this distinction, confessedly one of pathological grouping, curiously coincides with a real contrast of symptoms. Rarely or never have I met with the typical *nexus* of gastric symptoms just delineated, in concurrence with signs indicative of the presence of even moderate aggregations of tubercle in the lungs, much less of their disintegration and removal. Nay, more, even that very form of pulmonary tubercle most nearly allied to the condition of deposit which will be pointed out as commonly associated with gastric phthisis—the small, discrete deposits which, sown broadcast through the lungs, often, by their simultaneous ripening, set up acute phthisis[1]—appears rarely to approach in its gastric manifestations to the above symptoms.

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[1] From careful hospital researches, I have elsewhere (on the Medical Selection of Lives for Assurance, 3rd edition, p. 25) estimated that among phthisical patients of the middle classes, 35 per cent. of the relatives comprised in the four nearest degrees of blood-relationship (parents, brothers and sisters, uncles and aunts, and grandparents), are recognisably phthisical.

The febrile reaction of these cases constitutes another diagnostic mark, being greatly in excess of any such degree of this reaction as is ordinarily seen in ulcer of the stomach, much more of any merely functional derangement or dyspepsia of this organ. Taken in conjunction with the absence of considerable thoracic lesions, it really raises a strong presumption of the true nature of the malady. The general impression thus afforded may perhaps be illustrated by what are in some respects analogous offshoots of pulmonary tubercle: namely, cases of laryngeal phthisis. To subtract, from the characteristic *tout ensemble* of a case of laryngeal phthisis, the voice and breathing (including the cough) specific to the organ involved, would leave, as it were, very much the condition seen in many instances of gastric phthisis; in which the whole condition of the patient testifies to a deep-seated, intense, but yet latent irritation, such as even the marked gastric disturbance does but partially explain.

The characters of the pain itself are subject to even more than the usual variations affecting this symptom in the structural diseases of the stomach. In mere intensity, it rarely or never reaches that climax of severity which many patients term "spasm": but, within this somewhat vague limit, ranges through innumerable degrees, from weight and fulness to severe burning. Its situation shows a similar diversity; the epigastrium (the centre of which is its most frequent seat) by no means limiting its extent, and often failing to include its chief or only site. It sometimes spreads up behind the sternum, sometimes diverges towards both nipples; is sometimes referred to the umbilical region, sometimes exclusively occupies the space to the left (rarely to the right) of the navel. Tenderness almost always accompanies its worst forms and attacks; but is often transient, or is even found to be compatible with relief from very gentle, firm, and even pressure over the painful region.

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[1] Especially, as it seems to me, in the Celtic race.

In its relations to food, the pain shows another distinction from that of the structural diseases with which, in practice, this gastric phthisis is most likely to be confounded. Provoked by too bulky or too solid a meal, and often evidently called forth by some error of diet, it is yet much more capricious and uncertain in these respects than is, for example, the pain of gastric ulceration or cancer. Hence the instincts of the patient may often be studied and followed with much greater advantage; and, in all cases, improvement of symptoms may bring a corresponding improvement of dietary, with far less risk, and more rapidity. And as regards the period of digestion which is marked by the access of the pain, the preceding cursory description shows that this also is not fixed or characteristic; but is strictly one having a definite relation to the malady, quite as much as to the digestive act. In the outset of the symptoms, it is at the climax of digestive activity, or rather during its wane, when the pain is ushered in; a period which coincides less with any special copiousness of secretion, than with that powerful muscular action which wrings out the residue of the gastric contents through the reluctant pylorus.[1] Gradually the pain comes on earlier; until, at last, it is the deglutition, rather than the gastric contact of food, which seems to provoke it; much as the slightest noise or sound in his neighbourhood sometimes strings up a tetanic patient into agonized convulsion, or (to use a more homely illustration) much as an imprudent movement of the foot wakes up a dormant cramp in the leg.

The vomiting in these cases, though often quite as strictly the climax of the attack as it is in the similar paroxysm which is produced by the taking of unsuitable food into an ulcerous stomach (p. 117), varies widely from this relation in many well-marked instances. For in the more advanced stages of the malady, it occurs with such facility, and so rapidly after the ingestion of food, as to anticipate (if one may so say) all development of that paroxysm of pain which it thus displaces and substitutes. In rare instances we thus find the malady almost reduced to a single symptom, of which the true source or nature, evidently the chief point for clinical study, can only be made out with great difficulty and caution. For we have not only to guard ourselves against all the ordinary sources of such severe vomiting, but are at times obliged to remain in doubt whether the vomiting in the given case, even though essentially due to this gastric phthisis, is not increased or aggravated by some extraneous cause of this kind. Among such complications, hysteria, constipation, congestion of the liver, and ulceration of the bowels, are the most frequent and puzzling in actual practice—the two latter especially, from their frequent association with phthisis.

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[1] Compare p. 13.

The absence of hæmorrhage in gastric phthisis is one of those broad (yet safe) distinctions between it and ulceration or cancer of the stomach, which those who regard the practice of physic as based in great part on highly educated common-sense, are not likely to underrate or forget. Beyond a streak or so of bloody mucus, brought up, not in the vomit, but by the straining which attends it, and usually with its final efforts, I cannot recal a single instance of the association of hæmatemesis with a case of this kind—a circumstance which, if it be really the rule, certainly indicates that the stomach itself is rather bloodless than congested, or even enjoys almost an immunity from congestion during these attacks.

Constipation is the intestinal derangement commonly witnessed in connexion with gastric phthisis. Often, however, the bowels are unaffected. Diarrhœa is generally attributable to one of two causes: to the presence of some degree of phthisical infiltration or ulceration of the solitary and agminate follicles, on the one hand; or to hepatic congestion draining off, as it were, by this channel, on the other.

It is with reluctance that I append to the foregoing clinical sketch, which is little more than a summary of my notes of cases, any speculations as to the nature of the symptoms I have ventured to group under the heading of gastric phthisis. But, without attempting to define, scarcely even hoping to explain, its exact nature, I venture to point out those analogies which seem to me to indicate the true position of the gastric malady, and especially its relation to ordinary tubercle of the lungs. It is only by the study of such analogies that this mysterious group of symptoms seems explicable: only by the light reflected on these darksome recesses of the organism, from more exposed and accessible structures, that we gain any insight into the state of the stomach in gastric phthisis. Nor are attempts of this kind barren of general results. Both the science and the art of Medicine justify (and indeed demand) all reasonable effort to reduce every malady to its true position as a mere variation from the laws of health, rather than as a separate or superadded entity of disease.

If the foregoing outline of gastric phthisis be clinically accurate, it allows us to recognise, in the symptoms thus grouped, certain primary or essential disturbances of the healthy functions of the stomach. Of these deviations from health, pain, the most characteristic, is also the most vague; and implies little, save irritation of nerves. But that the sensibility (p. 44) proper to the organ is increased to a diseased extent, is much more significant; and, taken in connexion with the vomiting, which in its turn (p. 52) involves a wide and co-ordinate disturbance of the motor functions of the organ, shows that the irritation may be regarded as pervading the whole nervous apparatus of the stomach, or at any rate, both sensitive and motor nerve-elements of some part or parts of the organ. Nay, even the very time at which the paroxysm reaches its climax, points to the same conclusion; by showing that it is precisely the period of most energetic gastric digestion which is at first singled out for these abnormal acts of sensation and movement of the stomach.

As regards the secretion of gastric juice, there is abundant evidence that its quantity and quality are from the very first seriously affected; and that, in the more advanced and intense stage of the malady, the secretory act is reduced almost to a *minimum*. The clinical study of the vomiting, aided by a chemical analysis of the substances it ejects, scarcely admits of any other conclusion.

In the absence of any sufficient evidence of extreme congestion—or rather in the presence of that presumption against it which the rarity of any considerable hæmorrhage in these cases substantially amounts to—it is impossible to surmise whether to the irritation, exalted sensibility, and irregular muscular action, thus deduced from their symptoms, we are to add an undue vascularity of the mucous membrane of the stomach. But such a state is suggested by various analogies :—for example, by the analogous condition which Dr. Beaumont (p. 98) observed in the acute dyspepsia of intemperance, as well as by the analogy which seems to me (apart from any such suggestion) furnished by some forms of neuralgia, for the whole of that *nexus* of symptoms, to which the name gastric phthisis appears fairly applicable.

An ordinary face-ache seems, if carefully considered, to afford something like a clue to these cases : indeed, so much so, that there is a risk of finding too many, and too close, parallels between their circumstances, and thus of overlooking the wide diversity of nature which separates the two states. For such a face-ache, we may find two chief conditions : a segment of diseased nerve in some carious tooth, on the one hand ; and a disordered state of constitution, or of digestion, on the other. To open the mouth and eliminate the tooth, or to open the bowels and eliminate their peccant and superfluous contents, is what the educated common-sense of practical Medicine often regards as an alternative cure.

But the cure of this neuralgia, save in so far as it illustrates the connexion of remote parts in the malady, is of far less importance for our present topic than its elementary phenomena. Among these we may enumerate (1) excessive pain, ranging over a wide region of the adjacent tissues, but with a frequency and severity generally proportionate to the nervous relations implied by their supply from a common or congeneric division of the fifth nerve ; (2) undue sensibility, usually of still wider extent, and in the main, much more obviously and exclusively superficial or cutaneous in its situation ; (3) increased heat, vascularity, and interstitial fluid—the latter often amounting to enormous swelling of the whole of the cutaneous and subcutaneous tissues ; (4) muscular twitching and spasm, which though irregular in their date of occurrence, and comparatively subordinate in their effect, are yet sufficiently recognisable to be counted as tolerably constant ingredients of the malady.

Analogous elements to the foregoing are, I think, traceable in gastric phthisis. The irritated and carious dental nerve is represented by the irritated tissues and nerves of the diseased lung: the tubereulous deposit in which is shown by the common incident of hæmoptysis to disorganize tissues at least as large and resistant as nerve-tubes, long before it is sufficiently aggregated to be discernible to physieal examination in the living body. The deranged state of the constitution, or of the digestion, are also represented by corresponding conditions: which indeed, in respect of the function of digestion, are really often identical in the two neuralgiæ; in each of which errors of diet, both as to quantity and quality, or symptoms evidently tantamount to dyspepsia, precede and excite the larger disorder. Increased sensibility is, *mutatis mutandis*, no less distinet in the ease of the gastric mucous membrane than in that of the faeial integuments. Muscular spasm—which, as exemplified by tetanus and cramp, is mysterious as regards its relation with its causes, even beyond what is usual with abnormal nervous phenomena—certainly seems very diverse, if we merely contrast a twitching facial muscle, or the agonizing eramp of a gastrocnemius, with the co-ordinate aet of vomiting. Nevertheless, the maladies of the stomach and liver indicate (what it is scarcely necessary seriously to prove) that there is a certain connexion between irritation of the solar plexus of the sympathetie, and spasm of the unstriped muscular structures of the viscera it supplies, as well as with spasm of the striped voluntary muscles of the belly, and even of the limbs—a connexion such as no co-ordination having a merely final cause would apparently explain.

These cases of gastric phthisis may thus be regarded as a kind of neuralgia of the pneumogastric and sympathetic nerves:[1] certain thoracic segments of these conjoined nerves forming the starting-point of an irritation and morbid action, which is transferred to their abdominal connexions, and is manifested in the visceral branches of the solar plexus. It is quite possible that this latter irritation may substantially amount to a downright lesion of nerve-structure, and, through this, may bring about a lesion of gastric or allied tissues. It is certain that, in any case, the demarcation between functional and structural involvement of both nerve and organ is one which cannot at present be drawn; and which perhaps owes its very existence to the insufficiency of our means of observation. But, after all, this is no more than may be said for the most characteristic of the neuralgiæ: in which (as in many forms of rheumatism and sciatica) we may find in different cases every imaginable gradation between the completest evidence of local inflammatory action in the sheaths of the affected nerves and in the parts they supply, and a failure of all such evidence of lesion in connexion with the pain. That the former neuralgiæ are, as a rule, much more continuous or lasting, or are remittent, perhaps often regularly remittent, while the latter are more transient, irregular, and intermittent, are general rules, of clinical rather than of scientific import. But the neuralgia of gastric phthisis also shows indications of an approach, in various cases, towards one or other of these extremes: a fact the recollection of which may usefully remind us that sudden and great amendments at times occur independently of those dietetic or pharmaceutical measures to which the patient, or the physician, might otherwise be disposed to assign an apparent cure.

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[1] Two nerves more obviously, but not more essentially, connected in such a visceral neuralgia than in its facial analogue. Even as regards anatomical admixture, the difference is more of degree than of kind.

The treatment of these cases approaches, in so many of its features, that indicated by the ulceration of the stomach which they often closely simulate, that we may best consider it as contrasted with what has already (p. 165) been said in studying this lesion.

It may be premised that gastric phthisis is not only very amenable to systematic treatment (which, it is hardly necessary to say, must be distinguished from mere dosing with drugs), but that its worst symptoms often yield with a promptness very unlike the slower progress by which even the most favourable examples of ulceration of the stomach advance towards their cure. On the other hand, amendment is much more liable to be interrupted without any discernible cause; and, where it takes place, requires to be followed up by much more prompt and decided alterations of treatment, in order to prevent serious remissions or relapses. These characteristic features of its treatment are, I think, to a great extent explained by its neuralgic nature, and its relation to thoracic disease. But, however explained, the fact is scarcely to be doubted. I have repeatedly known the same patient who has been unable to take a teaspoonful of milk without vomiting, and has been extenuated by continuous pain, advance so rapidly toward amendment, and be subjected to such rapid enlargement of dietary during this process, as to be able in two or three weeks to consume a generous diet, containing (in addition to all the ordinary ingredients of a day's food and drink among the middle classes) one or two ounces of cod-liver oil; the patient meanwhile constantly showing a proportionate improvement in weight, colour, and bodily vigour.

Empirically speaking, opium and other strong sedatives are often but ill borne, still oftener unnecessary, in these cases; in which, on the whole, carbonates of the alkalies (either free or in effervescence with citric or tartaric acid) and minute doses of iodine, are generally of greatest service. Their benefits seem to depend, partly on their influence on the salivary secretion, and on the liver; partly on their neutralizing abnormal acidity (probably lactic acid from decomposition of starch and saliva) in the stomach; partly to the causes which, in the absence of any perceptible influences of this kind, seem to render these alkalies so serviceable in the earlier stages of pulmonary phthisis. Blistering is often of little avail; and, when made use of, may with advantage be transferred to any part of the thorax to which suspicion is pointed by local pain, or by auscultatory signs. Where there is already much feverishness, or heat of skin, it is better altogether dispensed with. Bismuth is often a valuable aid to the above drugs: especially when there has been any tendency to diarrhœa in the patient's previous history. The calumba, or some other light bitter infusion, is an excellent vehicle for the internal remedies: among which, by the way, prussic acid, or the sulphocyanide of potassium, deserve special mention. Mercurials in every form should be carefully shunned; even as occasional aperients, and in spite of the immediate relief they sometimes seem to bring. The simplest forms of purgative—castor-oil, tartrate of potash with rhubarb, rhubarb pill, extract of colocynth, guarded with henbane—are all that should be given by the mouth. And wherever there is the slightest suspicion (p. 178) of any accumulation of excrement in the cells of the colon—one might almost say whenever there is not strong presumption against such accumulation—a large gruel enema containing olive and castor-oil, or even assafœtida, should be administered. Such a procedure often brings about extraordinary and immediate relief to the gastric pain and vomiting which accumulation of this kind can always greatly aggravate, and of which, in some cases, it even seems to be the chief cause.

The various preparations of iron, though seldom well borne without careful preliminary treatment by some of the above remedies, are of great value. The effervescent citrate is perhaps the form in which they are best begun. The sulphate, as a pill, is also very serviceable. Both may be usefully combined with a minute (one grain) dose of the iodide of potassium.

As regards diet, the small and frequent doses of milk, or meat-broth, with farinaceous food, by which the ordinarily severe vomiting is best attacked, render this stage of the treatment closely akin to that of gastric ulcer. The absence of mastication must also be compensated in like manner.[1] Greater variety, and more rapid enlargement of the dietary are, however, to be early resorted to; and a moderate quantity of alcoholic stimulant (such as brandy, largely diluted with water or soda-water), is almost always beneficial from the very first. Rum and milk is also of great service in many instances. In others, however, it seems of absolute disservice—apparently from that great proneness to hepatic congestion which is discernible in many of the earlier stages of consumption, and which is certainly evinced by a very large proportion of these cases of gastric phthisis.

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[1] Insalivation, in some form or other, is so important a part of the digestive function, and the deglutition of unmasticated starchy food so injurious by the acidity and flatulence it is apt to cause, that, to a patient making use of this liquid diet, the homely expedient of sucking a small pebble, or chewing a bit of soft caoutchouc, is a remedy the suggestion of which needs no repetition to those who have experienced the relief it often brings.

LECTURE VIII.

On "Gout in the Stomach."

By the term "gout in the stomach," is usually understood an affection having its chief, if not exclusive, seat in the tissues of this organ; where it constitutes a manifestation of the gouty cachexia, sometimes preventing, but far more frequently replacing (by retrocession or metastasis) the ordinary paroxysm of articular gout. Whatever may have been the misgivings of some recent authors as to the frequency of this affection, or the chance of its being now and then assumed as the explanation of attacks more easily and directly referable to other causes, it is doubtful whether there is a single writer on the subject who does not either imply the above definition, or expressly affirm it.

There are many circumstances connected with the attacks thus named which, carefully weighed, go far to explain how "gout in the stomach" gradually gained this place in the literature of gout. The cases themselves are sufficiently rare, obscure, and diverse, to throw great difficulties in the way of their undergoing exact or frequent clinical comparison with each other. Their phenomena are essentially connected with symptoms, which are at least closely related to the morbid acts of the stomach (Chap. II.). They are not only extremely dangerous to life, but are sometimes fatal with a suddenness itself almost explaining the scanty or negative appearances met with in the subsequent necropsies. Lastly, their true import is only deducible from considerations, both clinical and necrological, which, until lately, have been scarcely at our disposal in respect of any of the organs they involve, especially in respect of the stomach. Taking into account that imperious necessity of an explanation which the striking character of the so-called "gout in the stomach" may fairly be said to offer, it may perhaps be regarded as having afforded until lately a perfectly legitimate and useful hypothesis: and it may even be questioned whether any one has a right to disturb it without being prepared to offer some more exact and satisfactory (if less exhaustive) explanation.

Conceding so much, it will not, I hope, be thought irrelevant, even in this condensed treatise on the diseases of the stomach, if I endeavour briefly to answer the question which years ago I used often to ask myself, and which since then has so often been pressed upon me by others—"What is gout in the stomach?" Rightly to answer this question would sum up the several answers to two branches of the interrogation. (1.) What do we see in actual practice suggestive of such an affection; and what is the exact nature of the cases thus grouped? And (2.) how far do these cases correspond with those descriptions of authors which represent what may be called our traditional knowledge of this affection? Without absolutely neglecting the latter and bibliographical branch of the inquiry, it is to the former that I shall chiefly address myself.

1. It can hardly be doubted that any sufficiently sharp or sudden dyspepsia occurring in persons known or suspected to be gouty is apt to be dignified by this term: a vulgar belly-ache taking rank, by courtesy, as gout in the stomach. Dr. Watson, in his admirable Lectures, has disposed of these pretentious ailments, in naming which, he suggests that "pork" should be sometimes substituted for "gout." That among the thousands of persons who, on any given day, are doomed by fixed fate or free will to eat something that disagees with them, and in whom the offending *ingesta* therefore excite gastric or intestinal disturbance, some should suffer severely; and that, in the ages and constitutions most exposed to gout, these sufferings should sometimes amount to downright danger—is not very surprising. For in many such persons the scales of life and death are always so nearly poised, that a very slight addition to the latter may easily determine its preponderance. And that irritation which provokes the salutary efforts of Nature to get rid of the *Trichinæ* or *Cysticerci* of underdone pork, or of the putridity of high game, and of stale fruit or vegetables which

have for days been partly infecting, partly disinfecting, a town-market, is such as may evidently form an efficient source of nervous exhaustion, as well as of the pain, vomiting, and purging it more directly provokes. It is hardly necessary to dwell upon the symptoms of such attacks, varying, as they do, according to the degree in which they have been provoked by undue quantity, or quality, of the *ingesta;* and according to the proportions in which indigestion or poisoning therefore take in the production of their symptoms. Even where most suggestive of gout in the stomach, all real claim to such a title is set aside by the most cursory study. Agonizing pain in the epigastrium, attended by nausea and enormous distension with flatulence, and relieved by eructation or vomiting, are symptoms which, occurring in a person previously or thereafter subject to gout, have often suggested such an explanation. But such symptoms also not infrequently attack persons whose youth, health (compare pp. 66, 296), and subsequent immunity from gout, completely negative any such view of their cause. Furthermore, if examined by the light of physiology and pathology, their phenomena constitute intrinsically evidence of nothing more than gastric irritation—irritation, too, devoid of any single feature suggestive of an arthritic origin. With no specifically gouty character, and no necessary (or even frequent) coincidence with the gouty habit, the most intense and mysterious cases of this kind are scarcely entitled to any deliberate scrutiny of their claims.

2. A less vulgar, though scarcely more valid, claimant to the title of gout in the stomach is to be found in the biliary cholic, which not only occurs with considerable frequency in the ages and constitutions most amenable to gout, but is caused by hepatic conditions closely analogous to those producible by gout, and—what indeed has greatly contributed to its having been awarded this title by able physicians—often so distinctly alternates with articular gout, as to suggest that it constitutes in some sense a displacement or metastasis of the disease from the joints to the stomach.

In the case of this biliary cholic, the sudden invasion of the disorder, the violent epigastric pain, the vomiting, the slow, laboured, irregular or intermittent pulse (more rarely quickened and thready), and the dyspnœa (or even asthma) which sometimes accompanies these abdominal symptoms, together evidently make up such a *nexus* of phenomena as closely corresponds with what is usually regarded as the description of gout in the stomach. But when these symptoms further occur independently of any distinct approach towards jaundice, or when (as a rarer but yet not absolutely infrequent occurrence) they replace, in an otherwise gouty individual, the impending or commencing fit in the joint, it is hardly to be wondered at that they should have received an interpretation referring them to a malady which (like the ordinary articular gout) is associated with a distinct structural lesion, and exhibits an intelligible train of symptoms, rather than to an affection like biliary cholic, many of the details of which we may truthfully say still lie almost beyond the pale of medical literature. What between the frequency of its occurrence, the diversity of its symptoms, the facility with which these are referred to the stomach, and the excessive difficulty with which a malady so vague, and so common, as well as so little dangerous to life, is traced to its proper necrological conclusions in the dead-house, there are probably few diseases which have hitherto so completely evaded full description and recognition as the biliary cholic caused by impaction of thickened bile in the hepatic ducts. And while the above circumstances—and among them the defects which have till lately attached to our knowledge of gastric disease—are greatly answerable for this want, there can be no doubt that the almost exclusive interest which has attached to what one may call the melodramatic aspect of biliary cholic—the paroxysm by which a gall-stone traverses the common duct—is also greatly chargeable with it. Even now, there are many otherwise able practitioners who scarcely recognise any biliary cholic devoid of jaundice; just as there are others who, unless they can locate the pain at or near that precise point which they are pleased to believe represents the contact of the gall-bladder with the wall of the belly, deduce, from the diverse and variable site to which the hepatic pain is often referred, a still more direct negative of its cystic or hepatic origin. And whatever be the accuracy of such strictures, it may be confidently asserted that much of what is called "gout in the stomach" is really unrecognized biliary cholic. Indeed, in some of the very cases which Scudamore especially adduces as typical examples of such gout, the biliary cholic may be recognised as complete *usque ad unguem*—that is, even to the very finger-nails of the patient, discoloured (as they must have inclusively been) by the jaundice which his accurate descriptions specify to have been present.

3. The source of confusion constituted by the presence of renal degeneration is one which it would perhaps be going too far deliberately to affirm as a frequent explanation of "gout in the stomach." But that disease of the kidneys, whether recognized or unsuspected, does account for some cases of this kind, it would be affectation to doubt.[1] And here, again, it must be owned that there is much to excuse such a diagnostic error. The gastric symptoms producible by renal degeneration—pain, vomiting, and prostration—are identical with those descriptive of gout in the stomach. Their invasion is often sudden: in some cases as marking the arrival of a stage or intensity of the renal malady, capable of sufficiently deranging the nervous system; in others, as connected with what is evidently a sudden check of renal secretion, such as is often due to corresponding renal disease, but is occasionally to be verified in complete independence of any structural lesion of the kidney. Furthermore, the attack is specially incidental to the gouty malady, and even to the gouty paroxysm, the renal degeneration being an element of the general disease. Indeed, it may be fairly asserted that some of these cases really are "gout in the stomach"—a genuine metastasis of the disease in so far as that it is the derangement or suppression of the articular paroxysm which occasions (or at least coincides with) the renal disturbance, itself provocative of the gastric and intestinal symptoms. But even granting all this, the term gout in the stomach remains quite inapplicable: alike to the whole symptoms; or to the gastric determination of urea, and the uræmia, which constitute their most striking and obvious phenomena; or to the renal disturbance which is their starting-point, and which—doubtful, as are perhaps its own claims, in strict-

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[1] An opinion for which, some years ago, I obtained an emphatic concurrence in conversation with the late Dr. Todd, who believed that much of the so-called gout in the stomach was attributable to this cause.

ness, to this title—is at any rate the only “metastasis” present. Besides, in the symptoms which usually accompany the ordinary paroxysms of such attacks, there is a good deal very diverse from the traditional “gout in the stomach.” Pain, for example, is rarely intense *and* local; in other words, if very severe, is for the most part spread over the whole belly, or even diverges to the head or limbs. Vomiting, again, is unduly prominent; and will be found to have generally been preceded by repeated attacks of nausea and retching, if not to have occurred long before the onset of the paroxysm. Purging, again, generally accompanies it; and is also, in many cases, a mere relapse or aggravation of a previous diarrhœa. The prostration of the organism generally, and of the circulation in particular, is also for the most part a gradual result of these symptoms, instead of being synchronous with their sudden invasion. Perhaps a similar caution may sometimes distinguish the epigastric pain which severe and repeated vomiting easily produces, from any such sudden and intense suffering as is ascribed to the access of gout in the stomach. On the whole, indeed, the resemblance is rarely close enough to justify any confusion of “gout in the stomach” with the perverted urinary effusion of these cases of renal disease: the less so, that a careful chemical and microscopical examination of the urine will of course generally assure their exact diagnosis.

4. Another probable claimant for some of these cases consists in that chain of phenomena which we occasionally see ushering in the close of valvular lesions of the heart, and which seem to be far oftener the mode of death where the heart is more intrinsically affected; either by degeneration of its muscular substance, or by disease of its coronary vessels, or lastly by a dilated and atheromatous state of that tube—the aorta—which measures and dictates its contractile force. As regards the mere symptoms of these cases, which in many respects approach the well-known characters of “angina pectoris,” it may be noted, that it is not so much in any special degree or site of the pain, nor even in the degree of vomiting, that their differentiation from the biliary cases above mentioned is to be sought for: but rather in the greater prominence or severity of dyspnœa on the one hand, and in the feeble, fluttering, thready

pulse, or threatened collapse, which testify to the more direct share of the heart in the paroxysm, on the other. Nevertheless as dyspnœa and irregular pulse are common attendants upon the biliary cholic of the aged, the practical distinction between the cardiac and hepatic varieties of the so-called "gout in the stomach" is sometimes by no means easy. The patient's face, the physician's first impression, are the safest grounds for a decision. And to discriminate between the laboured and oppressed state of the circulation in the one case, and its exhausted and prostrate condition in another, is a task which the most careful word-painting would scarcely help in. Doubtless, too, in some cases a certain degree of both lesions—cardiac and hepatic—is present simultaneously; just as either or both may be complicated by their renal counterpart.

In Dr. Gairdner's classical treatise[1] will be found some pithy allusions to the frequency and significance of the cardiac (and especially the aortic) lesions associated with inveterate gout. My own experience would lead me even further than he has gone: to clinical conclusions which, as regards the frequency of such lesions, quite correspond with the statistics of the dead-house; and, as regards their significance, affirm them to be often connected with few or slight symptoms, and to be compatible with considerable longevity. These propositions—that they are frequent, and in many instances of little influence on health—hold good, not only of their slighter and less developed forms, but even of a large proportion of those well-marked cases in which they could not possibly be overlooked by any one accomplished in physical diagnosis. So many of such cases figure in my note-books, and have been observed by me to enjoy tolerable health during long periods, and even to tide over severe illnesses, with little perceptible change of their physical signs, that I am almost afraid of under-rating their influence. Certainly when this is only known and guarded against, their prognosis, however cautious, ought on every ground to be anything but a gloomy one.

5. Is there any "gout in the stomach" left after the subtraction of these various affections? I can only say that I know of no such case: have never seen one: have never been able to

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[1] "On Gout," 4th Ed.

get trustworthy evidence of one from some of the most accomplished physicians living, or from the best records. Yet I would rather not deny its existence, save in the above qualified form. Just as, in acute gout and acute rheumatism, I have constantly found the stomach in such a state of tympanitic relaxation as suggests great irritation of the nerves of the organ, so I should surmise that it is quite possible there may be a more intense and exceptional form of this nervous irritation, productive of violent pain, vomiting, and cardiac disturbance, perhaps capable of giving rise to at least an hæmorrhagic effusion from the coats of the organ. That the dyspepsias of the gouty subject show a far-off approach to phenomena of this kind has already been stated; and, I may add, they are in this respect paralleled by the dyspepsias of convalescents from rheumatic fever, as well as (see p. 295) from ordinary fever. But such cases, as causes of death, must be infinitely rare. And while I should demur to their being accepted upon any evidence at present in the possession of scientific Medicine, I should question whether, if they occur, they would be rightly named "gout:" whether, in short, without having some specially or exclusively gouty origin, and gastric situation, they would be entitled to more than a place in those anomalous disorders of the nervous system, to which their phenomena seem so strictly to refer them.

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